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COMPLICATIONS OF OTITIS MEDIA WITHOUT RUPTURE OF THE MEMBRANA TYMPANI

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Complications of otitis media arise and all too frequently go undiscovered for want of a more careful study of the ear. Many a general practitioner has been misled by a failure to examine the ears in an effort to diagnose an obscure febrile condition. On the other hand, consultants are at times likewise guilty in failure properly to evaluate the significance of a slightly abnormal drum in the presence of an obvious intracranial lesion. The possibility of occurrence of surgical mastoiditis and other more serious complications of otitic infection without rupture of the membrana tympani, even without apparent involvement of the middle ear, has long been recognized. From my experience in one of the larger otologic clinics, it would seem as though the condition has, however, been frequently overlooked.

These atypical cases occur both in children and in adults. In the very young, the condition is called masked mastoiditis. In adults it has been variously described as atypical mastoiditis, primary mastoiditis, mastoiditis without apparent involvement of the middle ear, idiopathic mastoid abscess, etc.

Dabney,[1] in 1915, was probably the first in this country to become acutely interested in the problem of atypical mastoiditis, especially in those cases in which the membrana tympani was imperforate and in which there was no manifest evidence of otitic disease. He reviewed the literature, and among the comparatively few cases of atypical mastoiditis reported up to that time, he found twenty-four cases which authentically presented no rupture of the ear drum. He added two cases of his own. Since that time the condition has been recognized with increasing frequency, and a great deal of reporting of cases and discussion of them has obtained. It was first recognized in adults, and between the years 1918 and 1926, interest in this type of disease was at its height. The more or less inexact syndrome of masked mastoiditis in infants and young children was known after that time, and the literature to date now includes much in regard to otitic and mastoid

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1. Dabney, V.: Idiopathic Mastoid Abscess, J. A. M. A. **65**:501 (Aug. 7) 1915.

disease without apparent local symptoms, or definite anatomic changes, in infants, children and adults.

Macneil,[2] commenting on forty-five cases in children up to 2 years of age who were treated by operation at the Children's Hospital at Winnipeg during a period of two and one-half years, stated that 95 per cent were those of masked mastoiditis. He said:

> It is only within the past few years that masked mastoiditis occurring in infants and young children has come to be recognized. The number, considering the supposed rarity, would surprise one. The importance of a thorough otological examination in all cases of infantile gastro-intestinal disturbance combined with a rise in temperature and loss of weight, cannot be overestimated.

The detection of this condition seems to be dependent largely on the abnormal findings in the external canal and the membrana tympani. Babcock and Jones[3] said:

> The ear drum of masked otitis and mastoiditis is seldom red, and there is often little or no bulging. Spontaneous perforation is rare. Postauricular swelling, redness, and tenderness do not occur. However, there is always some discernible change in the drum or canal wall when carefully examined. Repeated myringotomies may be necessary to maintain middle ear drainage. Mastoid antrotomy is indicated (1) when repeated myringotomies for purulent otitis media fail to bring about improvement, (2) when the infant is markedly toxic in spite of free middle ear drainage, and (3) in the toxic infant when the middle ear suggests infection although myringotomy reveals no pus in the middle ear.

It is my experience that when myringotomy is done in typical cases of masked mastoiditis, an otorrhea usually does not obtain.

Campbell,[4] in reviewing some of the cases of severe otitic complications in adults during the epidemic of influenza of 1928 and 1929, stated: "These atypical cases always cause great difficulty in making a correct diagnosis, and are often overlooked until some more or less serious complications occur." He concluded that the existence of mastoid disease may occur without any obvious suppuration of the middle ear.

Warren[5] said:

> The infected middle ear, when draining by the eustachian tube or through a perforation of the tympanic membrane, can probably deal with the greater number of infections of the mucosa without the latter spreading to the underlying bone and causing caries. The mastoid antrum, on the other hand, which I believe is always

2. Macneil, F. A.: Masked Mastoiditis in Children Up to the Age of Two Years, Canad. M. A. J. **18**:688 (June) 1928.

3. Babcock, E. S., and Jones, J. R.: Masked Otitis Media and Mastoiditis in Infancy, California & West. Med. **31**:90 (Aug.) 1929.

4. Campbell, A. A.: Atypical Mastoiditis, with Report of Cases, Canad. M. A. J. **22**:201 (Feb.) 1930.

5. Warren, R.: Concealed or Unobserved Mastoiditis, Practitioner **127**:268 (Aug.) 1931.

infected in otitis media, with its thin mucosa and general inability to drain readily, is likely to remain infected, and should the aditus to the middle ear become blocked by oedema, polypi, etc., the infection readily spreads to the bony wall of the mastoid cavity, and thence to (1) neighboring parts such as the meninges, brain and lateral sinus, or (2) the system generally, as bacteremia or pyemia.

Other men, such as Coates,[6] Ersner, Kopetsky,[7] Phillips and Friesner,[8] Watson-Williams,[9] Eagleton,[10] Hempstead,[11] Hansel [12] and many others, have reported this type of case and discussed the differential diagnosis. None, however, so far as I have been able to determine, has stressed the possibility of occurrence of intracranial complications.

It is the purpose of this contribution to describe a group of cases which illustrate the pathologic possibilities in cases presenting only minor outward evidence of otitic disease, and to stress its frequency. Two most important facts will be emphasized: (1) there may be unrecognized extension from the middle ear to other portions of the temporal bone, such as mastoiditis and petrositis, and (2) of more serious consequence, intracranial complications of severe varieties may likewise occur.

ANATOMIC FACTORS; PATHOGENESIS

The tympanic antrum is the only pneumatic space present in the temporal bone of the new-born. At first it is filled with folds of embryonic tissue similar to granulation tissue, which is gradually absorbed. The absorption of this tissue is not complete until between the first and second years. The blocking of these spaces probably accounts for poor drainage from the antrum, and also for the delay in the rupture of the tympanic membrane in the early stages of the disease. Blocking of the aditus may occur during the progress of the disease after the condition in the middle ear has cleared up, and this accumulation of the infected material may be sufficient to give rise to focal symptoms in the absence of otitic symptoms, especially in young patients.

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6. Coates, G. M.: Mastoid Infection in the Infant, Ann. Otol., Rhin. & Laryng. **36**:913 (Dec.) 1927.

7. Kopetsky, S. J., and Almour, R.: Three Cases of Primary Acute Mastoiditis, Laryngoscope **35**:774 (Oct.) 1925.

8. Phillips, W. C., and Friesner, I.: Mastoiditis with Atypical Symptoms, J. A. M. A. **78**:1796 (June 10) 1922.

9. Watson-Williams, E.: Mastoid Disease Without Otorrhoea, Brit. M. J. **2**:1099 (Dec. 14) 1929.

10. Eagleton, W. P.: Brain Abscess from the Standpoint of the Otologist, Tr. Am. Acad. Ophth. **34**:64, 1929.

11. Hempstead, B. E.: Six Cases of Mastoiditis in Which the Middle Ear Was Definitely Not Affected, Ann. Otol., Rhin. & Laryng. **35**:517 (June) 1926.

12. Hansel, F. K.: Mastoiditis Without Apparent Otitis Media, Arch. Otolaryng. **3**:433 (May) 1926.

Gomperz [13] and Alexander [14] expressed the belief that the infantile membrana tympani is more resistant than the adult, and as the folds of mucous membrane in the middle ear and attic prevent the accumulation of a large mass of infected material, pressure on the drum with resultant necrosis and perforation is often much delayed. Alden [15] expressed the belief that the failure of the infant's drum to rupture early is more often due to the relief of pressure by an evacuation of the purulent contents of the middle ear by way of the auditory tube. Both factors probably play a part.

It is generally conceded that access of infection to the antrum in infants and to the mastoid in adults is gained from the nasopharynx through the auditory tube. In adults it passes from the middle ear through the aditus ad antrum, and then into the mastoid air cells. (It is possible to conceive of an infection of the blood stream localizing in the mastoid cells, but there is no reason to believe that it should do so there more than in the paranasal air cavities.) If the passage from the antrum to the mastoid cells is small, it is soon sealed off, leaving no means of drainage from those cells. As Portmann said, "The infection licks the middle ear and bites the mastoid." The infected middle ear drains through the auditory tube and clears up, hence the absence of great abnormalities of the drum membrane and subjective symptoms of otitis media. Roentgenograms in these cases in adults usually show a dense area around the antrum, indicating a more or less sclerotic or a very small cell partition between it and the mastoid cells, and the operative findings coincide with this finding. The case reported by J. M. Brown [16] illustrates this condition very well. Extension from the middle ear or mastoid intracranially may occur directly through intact bone, through areas of eroded and necrotic bone or by way of the blood or lymphatic vessels.

CLASSIFICATION

According to reported cases and to those that I have been able to study, in which the membrana tympani was not ruptured, the cases may be classified as follows:

1. Unrecognized acute otitis media and recognized or unrecognized mastoiditis.
2. Minor recognizable otitic symptoms and marked mastoiditis.

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13. Gomperz, quoted by Alexander.[14]

14. Alexander, G.: Diseases of Children, Philadelphia, J. B. Lippincott Company, 1917, vol. 6, p. 155.

15. Alden, A. M.: Mastoid Infection in Infants, Arch. Otolaryng. **5**:39 (Jan.) 1927; Gastro-Intestinal Disturbances in Infants as a Result of Streptococcus Infection in the Ears, South. M. J. **19**:360 (May) 1926.

16. Brown, J. M.: Atypical Mastoiditis: Case Report, Laryngoscope **34**:644 (Aug.) 1924.

3. Fulminating intracranial complications consequent to clinically unrecognized otitis media and mastoiditis.

(*a*) Single intracranial lesion the origin or nature of which is clinically unrecognized.

(*b*) Multiple intracranial lesions, some of which may be recognized or suspected.

Among the following reports will be found cases illustrating all of these eventualities.

REPORT OF CASES

CASE 1.—*Acute mastoiditis and labyrinthitis in a young man without rupture of the membrana tympani.*

Clinical History.—C. G., a man, aged 25, entered the hospital on Jan. 6, 1932, complaining of intense pain in the left ear and mastoid region, which radiated to the vertex and behind the eye, tinnitus, deafness and vertigo. Ten days before, the patient awoke with a severe earache and deafness on the left side. Three days after the onset, the pain occupied the whole left side of the head. Four days after that, the pains became sharp and more intense, and extended as far back as the occiput. For the past three days he had had spells of intense vertigo in which he had a tendency to fall backward and to the left. There was a history of mild otitis media in childhood with no recurrence since.

Physical Examination.—The patient appeared to be toxic, was pale and seemed to have some difficulty in maintaining his equilibrium, besides being in considerable pain. The right membrana tympani was normal. The left membrana tympani was of normal contour, but had lost its luster and was grayish, and the light reflex was distorted and almost absent. The superior portion of the canal wall near the drum was moderately edematous.[17]

There was a mild tenderness over the entire mastoid area on the left. The nose was normal, and the sinuses transmitted light well. The tonsils had been removed. Vestibular tests showed moderately hyperactive responses. Audiometer tests showed normal hearing for low tones and slightly decreased acuity for high tones, between C_5 and C_7. Roentgenograms of the mastoid showed the right side clear throughout; the mastoids were of the large pneumatic type. The left was greatly increased in density over the entire area, with evidence of absorption of bone in the region of the lateral sinus. The blood findings were: white cells, 5,800; polymorphonuclears, 82 per cent; lymphocytes, 16 per cent; eosinophils, 1 per cent; monocytes, 1 per cent. The temperature was 98.6 F.

Operative Findings.—A diagnosis of probable acute mastoiditis on the left side with toxic labyrinthitis was made, and myringotomy was performed. The discharge was minimal, and the culture showed no growth. The clinical condition did not improve, and on January 14, mastoidectomy was performed on the left side. The mastoid area was extensive and filled with granulation tissue and pus. The cell walls were completely broken down, and there was an erosion of the lateral sinus

17. Instead of the expression "sagging of the canal wall," it would probably be better to say that the portion described was edematous, and to describe the exact location and degree of involvement. The expression "sagging of the canal wall," so long used, suggests a displacement of the bony wall, or a drooping of the soft tissues due to a lack of tonus. The actual pathologic condition is an edema of the soft tissues due to a periostitis in that portion most nearly adjacent to the infection in the mastoid.

plate over an area 1 cm. in diameter. Culture showed hemolytic streptococcus. The vertigo disappeared gradually, and the patient was discharged from the hospital sixteen days later.

Comment.—There was very little external evidence of infection of the middle ear in this case. The left membrana tympani was only slightly gray and lusterless, and there was moderate edema of the superior posterior portion of the external canal near the drum. Myringotomy did not release any infected material from the middle ear, which was to be expected in this type of case, and culture at the time of myringotomy showed no growth. The mastoid cavity, however, was extensively necrosed.

CASE 2.—*Subacute mastoiditis of three months' duration in an adult, with only slight involvement of the middle ear and an intact drum.*

Clinical History.—E. B., a man, aged 32, presented himself at the clinic, complaining of intense pain in the left mastoid region, especially when opening the jaw. Three months before admission the patient had gone in swimming and had caught cold. A mild earache appeared several days afterward, which disappeared shortly. The earache recurred from time to time, however, and the past two weeks had become steadily worse. There was no otorrhea at any time, and there was no history of previous otitis media.

Physical Examination.—The patient was of massive physical build, and appeared to be in no great amount of distress. The right membrana tympani was normal. The left was red and thick, and the landmarks were entirely obliterated. The superior portion of the canal was definitely edematous near the posterior portion of the drum. The postauricular folds were barely absent, and there was only suggestive tenderness over the mastoid region. Roentgenograms showed the left mastoid greatly increased in density, with suggestive evidence of erosion of the lateral sinus plate. A blood count showed 8,400 white cells, with 80 per cent polymorphonuclears.

Operative Findings.—As a subacute mastoiditis was suspected, the left mastoid was opened and found to be extensive and entirely necrosed. There was a large area of erosion of the lateral sinus plate. Culture showed Streptococcus viridans. Convalescence was entirely uneventful, and the patient was discharged nine days later.

Comment.—This man had been observed and treated by a specialist for over two months, and although he did not suffer serious complications, it was quite evident that operative intervention should have been instituted sooner. A minute examination of the membrana tympani and the external canal would have put a close observer in mind of the existing condition immediately.

CASE 3.—*Acute mastoiditis in a young man, with an intact membrana tympani.*

Clinical History.—A. B., a man, aged 27, entered the hospital complaining of earache on the left side and pain and swelling in the mastoid region. The first symptoms had appeared four months previously, after he had been in swimming. He had a slight earache shortly afterward, and had had intermittent pain ever since. At no time was otorrhea present.

Physical Examination.—This patient was also of a large physical make-up, and of the swarthy type. He appeared to be experiencing only moderate discomfort. The right membrana tympani was normal. The left was gray, but not bulging, and was imperforate. There was no edema of the canal wall. There was tenderness over the entire mastoid region, especially in the region of the emissary vein. There was a slight edema over the mastoid area, with an obliteration of the postauricular folds. Roentgenograms showed the right mastoid large and clear throughout. The left was increased in density over its entire area, with quite definite evidence of absorption of bone in the region of the knee of the lateral sinus. The white blood count showed 15,000 cells, 79 per cent of which were polymorphonuclears, 77 per cent filament forms and 23 per cent nonfilament forms.

Operative Findings.—Mastoidectomy showed a large cell area with little destruction of cells except in the region of the knee of the lateral sinus, where a large abscess was found, with erosion of the plate. Simple mastoidectomy was performed, with removal of the lateral sinus plate. Culture showed Streptococcus hemolyticus. The patient was discharged from the hospital eleven days later entirely well.

Comment.—This case was almost entirely similar to the preceding one in that it started after the patient had been in swimming; the development was analogous; the duration was of the same length; culture was the same, and the operative findings were similar. This patient had had medical attention but twice, and was told that he had a catarrhal condition of the ear, of no probable consequence. It is easy to conceive of intracranial complications developing in both of these cases sooner or later, as erosion of the sinus plate was present and internal extension of the infection was on its way.

CASE 4.—*Acute mastoiditis with subperiosteal abscess without rupture of the membrana tympani in a young man.*

Clinical History.—G. M., a man, aged 22, entered the hospital complaining of earache on the left side and swelling and tenderness behind that ear. He had noticed a slight swelling behind the ear six days before, and since that time the swelling had increased. Pain commenced four days before admission, and was becoming more intense all the time. He had had a similar attack one month previously, after having been in swimming, but it subsided in one week without treatment. At no time was there otorrhea.

Physical Examination.—This patient seemed to be in considerable pain, in spite of the fact that nature was attempting to drain the abscess externally. The left membrana tympani was red and thickened, but not bulging, and the posterior superior wall of the canal was markedly edematous. There was a large, fluctuant, tender swelling behind the ear. Roentgenograms showed the whole postauricular area to be dense.

Operative Findings.—A diagnosis of mastoiditis with subperiosteal abscess was made, and the mastoid was opened. A large subperiosteal abscess was found. The mastoid area was of average size, and the cells were of the diplopneumatic type. There was a great amount of destruction just over the lateral sinus, with erosion of the plate. The vessel was covered with granulation tissue. The entire contents of the mastoid were exenterated, and the remaining portion of the lateral sinus plate was removed. Culture showed Streptococcus viridans. Recovery was uneventful, and the patient was discharged from the hospital eight days later.

Comment.—The diagnosis in this case was not at all difficult. It illustrates one course of drainage of an infected mastoid cavity that does not drain through the middle ear.

CASE 5.—*A fulminating, fatal case of acute otitis media and mastoiditis in a child. Membrana tympani intact.*

Clinical History.—A. M., a girl, aged 2 years, was admitted to the hospital with right-sided clonic convulsions. Prior to three days before admission, the patient was perfectly well. She had not had a sore throat, otorrhea or earache, and played normally. Three days before admission she ate breakfast and promptly vomited. She remained in bed the next two days; she was drowsy and refused to eat. For the past twenty-four hours she had had convulsions lasting about fifteen minutes, every half hour or so. They were clonic and involved the right upper and lower extremities only. Between convulsions she was stuporous and rigid, the legs were drawn up, and the head was held back in typical opisthotonos.

Physical Examination.—The right ear drum was normal, but the left was dull, and the landmarks were obliterated. The pupils were small and did not react to light. The Babinski sign was positive on the right; none was present on the left. Abdominal reflexes were absent. There were a paralysis of the left sixth nerve and a tendency to hold the head to the right.

Operative Findings. A diagnosis of encephalitis involving the left temporosphenoidal lobe secondary to mastoiditis was made, and the child was operated on soon after admission. The mastoid area was small. the dural plate was eroded, and the dura bulged into the mastoid cavity. Culture showed a nonhemolytic streptococcus. Death occurred three hours after operation, and necropsy was refused.

Comment.—This case illustrates the tremendous rapidity with which some of these cases can progress, especially in children. In all probability the process had been present for some time, but the symptoms were not noticeable until the last three days, when they appeared suddenly. These symptoms were undoubtedly due to intracranial extension of the infection, the exact extent of which cannot, of course, be stated.

CASE 6.—*Otitis media, mastoiditis, extradural abscess, abscess of the brain and venous sinus thrombosis in a middle-aged man, unrecognized until after death. No rupture of the membrana tympani.*

Clinical History.—J. W., a man, aged 44, was admitted to the hospital complaining of swelling of the nose and eyes. He said that five days previously the nose commenced to swell and became red, next the eyelids became red and swollen, and the right eye protruded somewhat. The patient was sent to the eye department first, where meningitic symptoms were recognized, and the neurologists were then consulted. There was no history of otitis media.

Physical Examination.—The patient was extremely ill when admitted to the hospital. There was a positive Kernig sign, the neck was rigid, and a right hemiplegia was present. The spinal fluid pressure was greatly increased, and the fluid was yellowish and turbid. The patient became irrational soon, and slept little. Drainage of spinal fluid and other supportive treatment were instituted, but the patient died four days after admission.

Autopsy Observations.—A large abscess of the temporal lobe and purulent meningitis were found. The dura over both petrous portions was intact and not

raised from the bone. The right membrana tympani was intact, and no discharge or evidence of the same was found in the canal. The right middle ear contained pus, and the mastoid area was entirely necrotic and filled with yellow pus. There was a bilateral retro-orbital abscesss. The cavernous, the right lateral and the inferior and superior petrosal sinuses on that side were completely thrombosed.

Comment.—This case was the termination of a long train of pathologic changes, beginning as otitis media on the right; then there was involvement of the mastoid, with extension and formation of an extradural abscess; there were an abscess of the brain and thrombosis of practically all the venous sinuses on the same side of the head. There was no history of the principal pathologic condition.

CASE 7.—*Right otitis media, antritis, right lateral sinus thrombosis and thrombosis of the straight sinus, the superior longitudinal sinus and, to a small degree, the left lateral sinus, in a boy of 17 months. Ear drums intact. Case diagnosed at autopsy.*

Clinical History.—G. S., a boy, aged 17 months, was brought to the hospital comatose and dehydrated, with a very rapid pulse and respiration. The father said that the patient was well until two weeks before, when he noted occasional twitching of the extremities and facial muscles. The child seemed listless and did not want to be handled. Hemiparesis set in on the left side five days before admission, and on the next day he had generalized convulsions. The last two days the patient was extremely lethargic.

Physical Examination.—The temperature on admission was 106 F.; the pulse rate, 130, and the respiratory rate, 36. There were a conjugated deviation of the eyes to the right and a fulness of the veins of the scalp and head, more on the right side. The optic disks showed a fulness of the veins of both sides, with hemorrhage on the right and blurring of the disk on the nasal side. A left spastic hemiplegia was present. The pupils were unequal, the right being larger than the left. The right membrana tympani was diffusely gray, and it was thought that the right ear might be the source of infection. An otolaryngologist, however, said he believed that the slight abnormality of the membrana tympani was of no consequence. A diagnosis of venous thrombosis of the veins of the right cerebral hemisphere was made. The patient died soon after admission.

Autopsy Observations.—Otitis media on the right side and antritis were found. There was a secondary septic thrombosis of the right lateral sinus with retrograde extension into the superior longitudinal sinus, the straight sinus and, to a small degree, the left lateral sinus. There was a hemorrhagic encephalitis of the right cerebral hemisphere due to the thrombosis of the cortical veins.

Comment.—This case illustrates graphically the possibility of intracranial extension of infection from the middle ear and mastoid antrum in a young child, in whom the initial source of infection was hidden. A significant feature was the fact that an otolaryngologist examined the ears and pronounced the slight changes in the membrana tympani of no significance. This case emphasizes the fact that in cases such as this one the slightest variance of the condition of the drum from normal should put one on one's guard.

CASE 8.—*Acute otitis media, mastoiditis and lateral sinus thrombosis in a 58 year old woman.*

Clinical History.—V. T., a woman, aged 58, was seen. The history obtained from the son was essentially as follows: For about four weeks she had been suffering from malaria. At first the chills occurred only every few days, but during the past two weeks she had had from one to three chills daily, each followed by a high fever. Previously the husband had been giving her quinine on the advice of a physician, a friend, who had not examined her. She also complained of pain over the left side of the face and ear, which was sharp at times, and most severe between chills. The husband said that she had been "nursing" the left ear for five or six months.

Physical Examination.—The day of admission she had a severe chill, with a temperature of 105 F., following which she vomited and then became unconscious, with a rigid body. The left membrana tympani appeared normal, with some loss of luster. The right was dull and gray, with no bulging, but there was an area of inflammation around the margin. There was a generalized spasticity of all extremities. A blood test showed: white cells, 6,000; polymorphonuclears, 90 per cent. The patient was examined and treated by many physicians for any symptoms and findings during her stay of one month in the hospital. Her renal condition received the most attention. She died one month after admission.

Autopsy Findings.—There were a bilateral otitis media without rupture of the membrana tympani, a bilateral mastoiditis and left lateral sinus thrombosis with extension low in the jugular vein. There were an abscess of the left kidney and a cervical cellulitis with abscess formation on the left side. Culture showed Streptococcus haemolyticus.

Comment.—In this case the primary disease was entirely overlooked, even though the accurately described findings in the middle ear and the external canal pointed so definitely to the real condition. Lateral sinus thrombosis, mastoiditis, metastatic abscess formation and septicemia developed, and the patient died.

CASE 9.—*Left otitis media, mastoiditis, lateral sinus thrombosis and meningitis in a boy, without rupture of the membrana tympani.*

Clinical History and Findings.—H. C., a boy, aged 17, came to the ear clinic complaining of pain in the left ear, which had been present for one month. The right membrana tympani was comparatively normal, except for a slight loss of luster. The left was reddened in the region of the handle of the malleus, with slight retraction. The left drum was lusterless, and there were an abnormal reddening and edema of the superior portion of the canal wall near the drum. The pharynx was inflamed, and the tonsils were septic. A diagnosis of left catarrhal otitis media was made by an otolaryngologist, and only local treatment was instituted. Two months later the patient was seen in the outpatient medical clinic with a complaint of severe frontal headache. He was toxic, looked anemic and generally was acutely sick. The history at that time was briefly as follows: He had had pain in the left ear for three months, headache on the left side for one and one-half months, loss of weight and appetite, anorexia and nausea for the past eight days, vomiting for the past three days, drowsiness for the past two days and periods of irrationality and coma the preceding day. The patient had never had a discharge from the ear. Roentgenograms of the mastoids showed the right one

to be clear, and the left mastoid was greatly increased in density. Culture of the spinal fluid showed a growth of Pneumococcus.

Operative Findings.—A detailed neurologic examination pointed to the existence of an abscess of the left temporal lobe. The patient was operated on through two trephine openings, and no abscess was found. There was evidence of intracranial pressure, however. The patient died shortly afterward.

Autopsy Observations.—Otitis media and mastoiditis on the left side, lateral sinus thrombosis and a purulent meningitis were seen. The left mastoid cavity was entirely necrotic.

Comment.—This patient first presented himself in the otologic clinic with a history of earache on the left side and pain in the mastoid region which had been existent for one month. The otolaryngologist who examined him recorded the findings accurately, and yet failed to evaluate them in the light of the greatest pathologic possibilities. The disease progressed to intracranial complications which no treatment was adequate to combat, i. e., lateral sinus thrombosis, abscess of the brain and purulent meningitis.

COMMENT

That otitis media and its complications may occur without rupture of the membrana tympani is not a new conception. Since 1915, when Dabney [1] made a comprehensive analysis of reported cases up to that date, it has been recognized, and many workers have described and discussed the condition. I believe, however, that the fact that it is still frequently overlooked, or its significance underestimated, is worth while considering. The condition is much more frequent in children, because although the focus of infection is smaller in extent than in the adult, the systemic reactions are more labile, and severe constitutional reactions occur in the presence of infection in the middle ear and antrum before the drum has had time to perforate, which would not obtain in the adult.

The cases of unrecognized otitis media and mastoiditis that do not exhibit definite or severe symptoms present the greatest difficulties in diagnosis, and consequently are the most dangerous from the standpoint of intracranial complications. The patient in case 9, for instance, presented only minor changes in the membrana tympani and canal, and there was no tenderness over the mastoid area or other signs suggestive of extensive disease. The patient was assured that his disease was of minimal consequence, and it was not until two months later that he again sought medical aid. By that time the complications of lateral sinus thrombosis and purulent meningitis had occurred, and even the most heroic treatment availed nothing.

Cases that present recognizable but minor otitic symptoms and marked mastoiditis are of course not so difficult to diagnose, especially when there are roentgenographic findings. They should not be temporized with, however, once a diagnosis has been made. Myringotomy

does not alleviate the conditions, as most of them have been existent for a considerable length of time, and drainage through the middle ear is not feasible, or it would have drained this way before. Cases 2 and 3 illustrate this type well. Although there were only minor findings in the middle ear, the mastoiditis was far advanced and was presenting severe symptoms. Mastoidectomy gave prompt relief.

In the cases presenting fulminating intracranial complications when the otitis or mastoiditis has not been recognized, the intracranial lesion may or may not be definitely localized and recognized. In these cases a most painstaking study of the ear and mastoid is necessary. In case 6, the meningitis was recognized and treated, but the origin of this and the abscess of the brain and extensive sinus thrombosis were not suspected. In case 7, intracranial complications of otitic origin were suspected, but the otologist assured the neurologists that the slight abnormality of the right membrana tympani was of no consequence, even though a diagnosis of venous sinus thrombosis on that side was quite definitely made. The condition of the right membrana tympani, together with the chills, septic temperature and metastatic abscesses, should have suggested infection of the middle ear and the mastoid. The true seat of the disease, however, was overlooked for a full month, and then was not recognized until autopsy.

The causes of failure in the diagnosis of these obscure conditions seem to have been due to (1) an insufficient history, (2) a failure to associate a distant manifestation with a possible otitis media, (3) a lack of proper evaluation of minor anatomic changes in the external ear and membrana tympani and (4) an insufficient follow-up and close observation of cases presenting minor otitic changes in which dangerous potentialities for complications exist.

Minute examination of the ear canal and drum is all important, and any deviation from the normal should be viewed with suspicion. It is important in cases in which doubt arises to exhaust every available diagnostic measure in order to come to an accurate conclusion. Complete study of the blood, roentgenograms and hearing tests should be made in every case when possible. Myringotomy should be done as a diagnostic measure more than as a curative one, and a culture should be made. One must not be misled by the fact that free drainage does not follow myringotomy, and that the culture may not be obtained (case 1 illustrates this eventuality in an adult). It must be borne in mind that in these cases the disease in the middle ear is minimal if existent at all, which accounts for the ear drum not being perforated in most cases. When infection in the adult mastoid or infantile antrum is proved, or even strongly suspected in these obscure cases, with or without intracranial complications, not only is operation justified, but failure to operate is clearly temporizing with a serious condition.

CONCLUSIONS

1. Otitis media and mastoiditis with intact membrana tympani occur more frequently than is generally supposed.

2. Infection may extend from the middle ear to the mastoid and intracranially without gross pathologic changes in the middle ear.

3. Streptococcus haemolyticus is probably the most frequent and most dangerous bacterial agent in the most severe cases.

4. Minute examination of the ear canal and drum is all important, and any deviation from normal should be viewed with suspicion.

5. It is important, in cases in which doubt arises, to exhaust every available diagnostic measure in order to come to an accurate conclusion.

6. The condition is more common in children than in adults.

7. The causes of failure in diagnosis are: (*a*) an insufficient history; (*b*) a failure to associate a distant manifestation with a possible otitis; (*c*) a lack of proper evaluation of minor anatomic changes in the external canal and the ear drum; (*d*) an insufficient follow-up of potentially dangerous cases.

8. In this section of the country swimming is a frequent cause of this type of disease.

9. The expression "sagging of the canal wall" should be deleted from otologic usage, and the term "edema" substituted, which describes accurately its location and extent.

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CONTINUOUS STIMULATION OF THE LABYRINTH WITH SUSTAINED NYSTAGMUS

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In previous labyrinthine studies wherein caloric stimulation was employed, one of us (Spiegel) observed that reflex movements of the eyes still appeared even though the stimulus had lasted for several hours. As this fact seemed to shed some light on the mechanism of the caloric reaction of the labyrinth, a more detailed study of this phenomenon seemed worth while.

METHOD

To stimulate the labyrinth we constructed a simple apparatus by means of which water from a flowing faucet could continuously flow through tubes to the labyrinth and back again to the drain. At the end of the rubber tubing connecting with the animal, a small U-shaped brass tube was placed which consisted of two small brass tubes soldered side to side, each 7 cm. long and 2 mm. in external diameter. The ends that were to project into the auditory canal were closed by a brass cap. The common wall of both tubes was excised for a short distance near the cap, thus permitting water to flow freely from one tube into another. In one series of experiments, this U-shaped apparatus was introduced into the external ear, and in another group the ear drum was perforated, and the apparatus introduced so far into the middle ear that it touched the median wall. The water used for this stimulation, when leaving the apparatus, was usually at a temperature of 17 C. The maximum temperature used for stimulation with cold water was 23 C.; the minimum temperature was 8 C. To reach temperatures below 17 C., it was found necessary to conduct the water from the supply pipe through a coil of brass tubes immersed in an ice-salt mixture before its induction into the apparatus.

In some experiments, the caloric responses were elicited with hot water (50 C.). In another group, stimulation was accomplished by galvanic current, which will be spoken of later in the article. The experiments were performed on cats kept under superficial ether anesthesia in as uniform and constant a degree as possible. Special care was taken to maintain the head rigidly in the desired position during the experiment.

RESULTS

The latent period for the appearance of nystagmoid movements in the eyes, on stimulation with cold water, was in some cases very short, from about ten to twenty seconds, while in other cases it was observed

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that several minutes elapsed before such movements were noticed. As the particular interest in this work was in the nystagmus induced after a prolonged stimulation, the conditions that influenced the length of the latent period were no longer analyzed. The nystagmoid movements do not usually continue without interruption, but periods of rest alternate with periods in which the movements reappear (figs. 1,

Fig. 1 (cat Cal. 11).—July 13, 1932. Continuous stimulation of the right ear (temperature of outflowing water 19 C.). Movements of the right eye. The stimulation started at 5:10 p. m. This section of the graph was recorded at 5:40 p. m. The upper line indicates the time in seconds.

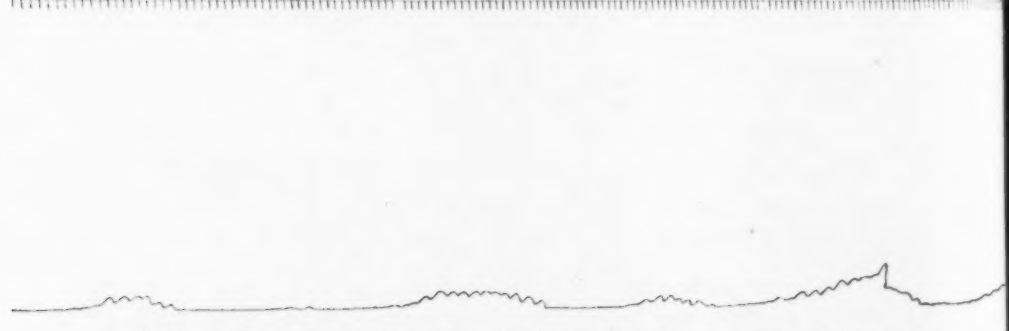

Fig. 2.—The same experiment as in figure 1 continued. This section of the graph was recorded at 6:10 p. m. (stimulation lasted one hour).

2 and 3). These periods of rest last from ten seconds to over one minute. To study these alternating periods of rest and excitation of the labyrinthine reflex arc in detail, movements of the eyes were recorded on a kymograph. A silk thread was fastened on the sclera at the insertion of the external rectus muscle and connected with a very light lever. A study of these graphs showed that in some cases, at least for a certain time during such continuous stimulation, the nystag-

moid movements appeared in groups of about equal duration, and that the lengths of the pauses between these groups were also approximately equal. This phenomenon is particularly striking, if the periods of rest become relatively long and the nystagmoid movements appear in forms of attacks (fig. 3). During such attacks the animals become much more excited, probably on account of dizziness.

The movements of the eyes were, as a rule, horizontal or mixed, with the fast component toward the opposite side, as long as the head was held in the normal position. Occasionally a few vertical movements were noticed between the horizontal ones.

A question arose as to whether the nystagmus observed, even after several hours, could be explained by the usual theories of caloric stimulation. The assumption that the part of the endolymph first affected by syringing (caloric stimulation) becomes heavier and sinks down is still the most accepted and best founded. The measurements of Frenzel [1] and particularly of Schmaltz and Voelger [2] and Schmaltz [3] showed,

Fig. 3.—The same experiment as in figures 1 and 2 continued. This section of the graph was recorded at 6:45 p. m. (stimulation lasted one and three-fourths hours).

indeed, that the temperature on the wall of the horizontal semicircular canal is changed by syringing the meatus externus, and the experiments of Maier and Lion [4] make it probable that an endolymph movement is produced by the calorization.

Recently, W. Steinhausen [5] was even able to observe the deviation of the cupula in the labyrinths of pikes after heating the semicircular canal. In our case, the question arose as to whether sustained calorization can produce a continuous movement of the endolymph and thus a continuous stimulation of the end-organs. A priori, one might think that a fluid contained in a closed system of tubes and cooled by lowering the temperature of a certain small part of its wall shows a flow only until all parts of this fluid have reached the same low temperature. As the quantity of fluid contained in the membranous labyrinth is rela-

1. Frenzel, H.: Arch. f. Ohrenh-, Nasen- u. Kehlkopfh. **113**:233, 1925.
2. Schmaltz and Voelger: Arch. f. d. ges. Physiol. **204**:708, 1924.
3. Schmaltz: Arch. f. d. ges. Physiol. **208**:324, 1925.
4. Maier, M., and Lion, H.: Arch. f. d. ges. Physiol. **187**:47, 1921.
5. Steinhausen, W.: Arch. f. d. ges. Physiol. **228**:322, 1931.

tively small, one would expect that the adjustment of differences in temperature between the different parts of the endolymph is reached within a short time; however, the fact that nystagmus can still be elicited if the calorization is continued for from one to two hours is contradictory to such an assumption. The question arises whether the continuous application of low temperature on the external meatus or on the external and middle ear does not finally reduce the activity of the nervous apparatus of the inner ear by a directly paralyzing influence of the low temperature, or by inducing changes of the circulation in the inner ear.

In regard to the usual method of short-lasting syringing, particularly the experiments of J. Kubo,[6] de Kleyn and Storm van Leeuwen [7] have shown that the theory of Bartels [8] cannot be held; this theory ascribes the effect of stimulation with cold water to a paralysis of the end-organ. These authors showed, in their experiments on animals, as Bárány previously had demonstrated in man, that changes of the posture of the head could produce a change in the direction of the caloric nystagmus. This is easily explained by Bárány's theory, as the change of the position of the head changes the relation of the ampulla to the remaining semicircular canal, and thus stimulation with cold water can produce in some cases an ampullopetal, and in others an ampullofugal, movement of the endolymph. In spontaneous nystagmus due to paralysis of the labyrinth, such changes in the direction of the nystagmus, depending on the position of the head, are not observed.[9]

Although the aforementioned experiments of Kubo and of de Kleyn and Storm van Leeuwen are of the greatest value for an understanding of the nystagmus in the usual short-lasting calorization, one cannot draw conclusions from these experiments regarding the nystagmus observed in long-standing caloric stimulation of the labyrinth.

Thus, how far the nystagmus observed in continuous calorization is due to an endolymph movement, or how far it might be ascribed to a paralysis of the end-organ, had to be studied. To ascertain whether the continuous calorization is able to paralyze or suppress the tonic impulses sent by a normal labyrinth toward the central nervous system,

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6. Kubo, J.: Arch. f. d. ges. Physiol. **114**:143, 1906.

7. de Kleyn, A., and Storm van Leeuwen, W.: Arch. f. Ophth. **94**:316, 1917; **107**:109, 1922.

8. Bartels: Klin. Monatsbl. f. Augenh. **50**:200, 1912.

9. One might think that change of direction of the caloric nystagmus in different positions of the head might have to do with the otolithic apparatus, but even after separating the otoliths from the maculae by centrifugation in guinea-pigs, the changes of position of the caloric nystagmus were typical in different positions of the head, according to de Kleyn and Lund (Acta oto-laryng. **6**:92, 1922).

one labyrinth was destroyed, and the effect of the continuous calorization of the normal side on the spontaneous nystagmus was studied.

CAT Cal. 7. 3/4/32. 11:00 a. m. Right labyrinth was extirpated, nystagmus to the left ++.

12:20 Rate of nystagmus 66 per minute
12:30 Rate of nystagmus 62 per minute
Continuous calorization of the left ear begins (apparatus in middle ear).
12:35 Rate of nystagmus 26 per minute
12:38 Rate of nystagmus 28 per minute
12:44 Rate of nystagmus 36 per minute
1:00 Rate of nystagmus 36 per minute
1:10 Rate of nystagmus 32 per minute
1:20 Rate of nystagmus 22 per minute
Calorization discontinued.
3:50 Rate of nystagmus 70 per minute
4:00 Rate of nystagmus 72 per minute

One sees that continuous calorization of the normal labyrinth can lower the frequency of spontaneous nystagmus that follows extirpation of the opposite labyrinth, but that it is unable to suppress this spontaneous nystagmus. One might conclude that by continuous calorization one can lower the function of a normal labyrinth, but not really paralyze it.

First of all it seemed necessary to study the influence of the position of the head on the direction of this nystagmus in continuous calorization of the labyrinth. Let us mention first such cases in which the apparatus was introduced only into the external ear.

CAT Cal. 9. 3/31/32. Apparatus in right meatus externus.
Temperature of the outflowing water 14 C.

3:35 Continuous outflow begins; normal position of the head
3:45 Nystagmus horizontal to the left
4:02 Nystagmus horizontal to the left; rate, 5 per minute
4:08 Identical
4:21 Apparatus removed
Four rods are fixed upright on the edges of the animal board, and the board turned over 180 degrees so that it stands on the rods and the animal is suspended with its back to the table
4:34 Apparatus introduced in the right meatus externus again; the outflow begins
4:35-4:37 Nystagmus to right side
4:38 No nystagmus
4:40 Some nystagmoid movements to right
4:48 Nystagmus to right
4:53 Nystagmus to right; rate, 5 per minute

We find here that a change of position of the head causes a reversal of the nystagmus. As long as the animal and the head are in the normal position, the nystagmus is directed toward the nonstimulated side; when

the animal is turned 180 degrees and lies on its back, the nystagmus is directed toward the side of stimulation as in the experiments of Kubo and of de Kleyn and Storm van Leeuwen, in which calorization was of short duration.

The same was observed in some cases in which the apparatus was introduced into the middle ear.

CAT Cal. 10. 4/7/32. Drum perforated.

11:09 Apparatus introduced into the left middle ear; animal in normal position; outflowing water 8 C.; nystagmus to opposite side starts almost immediately

11:20 Animal lying on back; continuous outflow; temperature of the outflowing water 15 C.

11:27 Nystagmus distinctly to left side; groups of nystagmoid movements, interrupted by periods of rest of nearly one minute's duration

11:33 Nystagmus to stimulated ear

11:39 Nystagmus continues

11:40 No nystagmus

11:42 Some single nystagmoid movements toward stimulated ear

11:45 More frequent nystagmus to stimulated ear

11:45-11:46 Rate of eye movements, 7 per minute

11:49-11:50 Rate, 21 per minute

11:55 Nystagmus continues

11:58 Animal again in normal position; nystagmus to right side (to the nonstimulated ear)

12:00 Animal vertical, head downward

12:00-12:07 Direction of nystagmus uncertain; only a few eye jerks

12:08 Head upward; animal vertical; distinct nystagmus to right side; rate 30 per minute

12:10 Animal vertical, head downward

12:12 Direction of nystagmus not quite certain (questionably to right); rate of eye movements, 9 per minute; outflowing water, 22 C.

We find here, again, that the nystagmus is directed toward the normal side in the normal position of the head and of the animal, the same being observed in the vertical position with the head upward. When the animal lies on its back, the nystagmus is directed toward the side of stimulation. So far the observations are in accordance with those of Kubo and of de Kleyn. When the animal was held vertically with the head downward, the direction of the few eye jerks was not quite certain. Some seemed to be directed to the nonstimulated ear. This would not correspond with the observation of the aforementioned authors, but the direction was not sure enough to draw definite conclusions. In any case, we see here, again, that in the horizontal position of the animal, lying on its back, the direction of the nystagmus is reversed.

In some cases (cat 4, temperature of the outflowing water 18 C.) such a reversal of the direction could not accurately be noticed, whereas in the normal position (animal horizontal, lying on abdomen) the nystagmus was directed to the nonstimulated ear. No certain difference between both components of the nystagmus could be detected in the other positions.

Finally, however, some cases could be observed in which the direction of the nystagmus was not influenced by the change of the position of the head.

CAT Cal. 8 3/31/32. 11:00 a. m. Apparatus in the left middle ear; outflow 19 C. Normal horizontal position, animal lying on its abdomen; nystagmus to right side
12:35 Animal lying on its back; nystagmus to right side
12:40 Outflow stopped
12:41 Continuous stimulation with hot water (50 C.); nystagmus to the side of stimulation; some vertical movements to the upper lid
12:50 No nystagmus
12:51 Nystagmus starts again
1:10 Some few nystagmoid movements for a minute
1:15 Animal lies on abdomen; nystagmus more frequent and is directed toward side of stimulation; rate, 16 movements in thirty seconds

Let us consider first those cases in which an influence of the position of the head on the direction of the nystagmus was noticed during continuous calorization. This would indicate that in these cases an endolymph flow was the cause of the ocular reaction, although the calorization was continued for a longer time. But is it possible that continuous calorization produces more or less a continuous endolymph flow? It was pointed out that the endolymph movement produced by cooling of the wall of the horizontal semicircular canal should discontinue as soon as all parts of the fluid reached the same temperature, but there exists a possibility that the temperature of the endolymph is increased again by the surrounding perilymph fluid or by the blood in the contiguous capillaries. Hence, these cases of continuous calorization could be understood from the point of view of Bárány's theory, if the influence of such factors which try to increase the temperature of the endolymph is considered.

This consideration, however, cannot explain the group in which the direction of the nystagmus is not influenced by the change of the position of the head. Here one has to assume that the change of temperature finally can impair the function of the end-organ by a direct paralyzing action of the low temperature or indirectly by changes of the circulation (production of vasoconstriction).

It was mentioned that the nystagmus in continuous caloric stimulation appears, at least in some cases, in more or less regular groups of

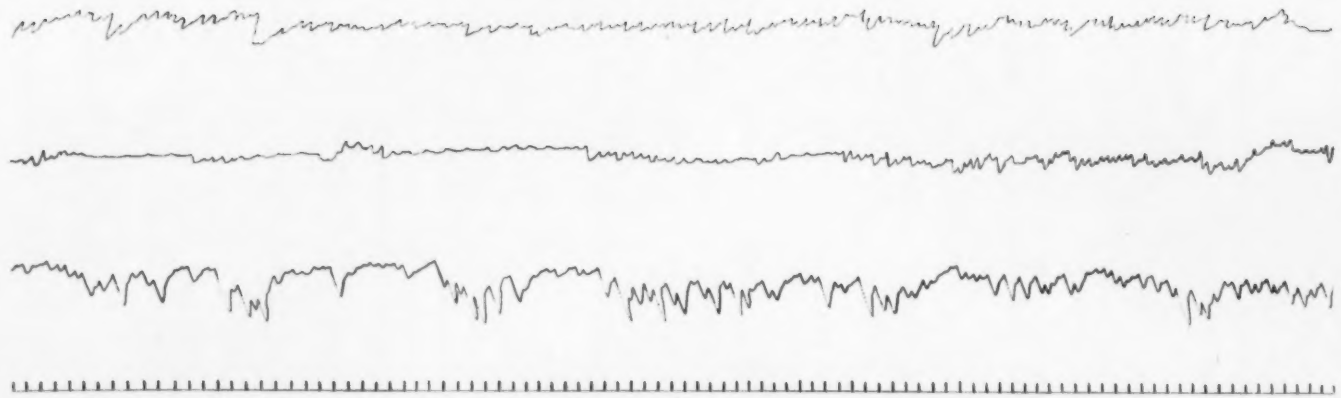

Fig. 4 (cat 13).—July 4, 1932. Galvanic stimulation. Anode in the left external auditory meatus. Stimulation started at 3:47 p. m. Stimulating current, 15 milliamperes. Movements of the right eye recorded. The uppermost graph was recorded at 4:12 p. m.; the middle graph, at 4:45 p. m.; the lowest graph, at 5:02 p. m. The time is in seconds.

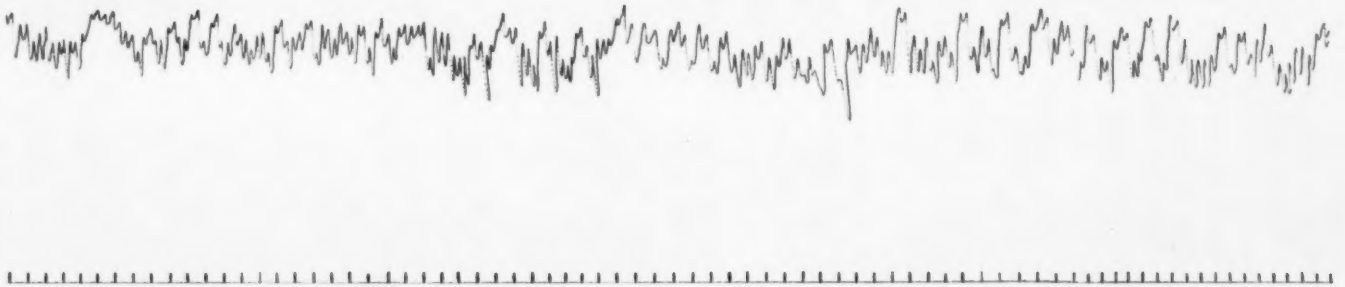

Fig. 5.—The same experiment as in figure 4 continued. This section of the graph was recorded at 4:48 p. m.

ocular movements (figs. 1, 2 and 3). This observation requires further elucidation. One might conjecture that the endolymph in such cases does not flow continuously with uniform speed, but the speed of its flow increases and decreases rhythmically. Such a hypothesis is not applicable to those cases in which the direction of the nystagmus does not depend on the position of the head and in which the nystagmus has to be explained by a paralysis of the labyrinth.

Hence, the question arises as to how the labyrinth reacts to any other form of continuous stimulation. To answer this question, the labyrinthine reflex arc was stimulated in a group of cats by a constant galvanic current for several hours. The animals were kept under superficial ether narcosis, which was as uniform as possible. The stimulating electrode was introduced into the external auditory meatus, the indifferent electrode being applied to the shaved abdomen. The movements of the eyes were similarly recorded on a kymograph, as in the above mentioned experiments. In this form of stimulation, which is much more intense than stimulation with cold water, total cessation of ocular movements was not observed as in calorization. A study of these graphs showed, however, a rhythmic increase and decrease of the movements of the eye, especially after some duration of the stimulation (figs. 4 and 5). One must assume that in this galvanic stimulation likewise there are a wavelike increase and decrease of excitability of the labyrinthine reflex arc.

In former experiments Spiegel and Yaskin [10] found that normally tonic stimuli from the resting labyrinth to the central nervous system also show rhythmic waves. In these experiments spontaneous nystagmus after extirpation of one labyrinth, arising from normal impulses from the remaining normal labyrinth, showed periodicity of rest and excitation. Even Bechterew's nystagmus, which appears on extirpation of the second labyrinth, after compensation of the loss of the first labyrinth, had such a periodicity in the experiments of Spiegel and Yaskin. The origin of the impulses producing Bechterew's nystagmus should be sought in the vestibular nuclei on the side of the extirpation of the first labyrinth, as shown in the experiments of Spiegel and Démétriades.[11] As the graphic record of Bechterew's nystagmus showed a periodic increase and decrease of ocular movements, similar to the graphs in extirpation of one labyrinth, one must conclude that not only the peripheral labyrinth, but also its centers in the rhombencephalon, react intermittently to normal endogenous stimuli.

10. Spiegel, E. A., and Yaskin, J.: Ztschr. f. d. ges. exper. Med. **63**:515, 1928.
11. Spiegel, E. A., and Démétriades, T. D.: Arch. f. d. ges. Physiol. **210**:215, 1925.

In the present investigation it was found that the labyrinthine reflex arc likewise reacts to artificial external stimuli, such as calorization and galvanization, with a tendency toward periodicity. Theoretically, it is possible that the lymph flow produced by calorization might increase and decrease in its speed. Such an assumption, however, is seemingly unnecessary to explain the appearance of nystagmoid movements in groups, because these phenomena can be understood by the fluctuations in excitability of the labyrinthine reflex arc itself. It is interesting to note in this connection that recently, Brody and Dusser de Barenne [12] have obtained wavelike fluctuations in excitability in carefully executed stimulation of the cortex cerebri in cats under constant conditions. There is a possibility that our observations of fluctuations of excitability of the labyrinthine reflex arc and those of Brody and Dusser de Barenne on the cortex are expressions of the same underlying intrinsic factors.

SUMMARY

1. Continuous caloric stimulation of the labyrinth can produce nystagmoid movements of the eye, which appear, interrupted by short periods of rest, for several hours. Spontaneous nystagmus following extirpation of one labyrinth is lowered in its frequency by continuous stimulation of the opposite labyrinth with cold water.

2. The direction of this nystagmus is, in the majority of cases (calorization of the external or of the middle ear), influenced by the position of the head, but in some cases of calorization of the middle ear such an influence of the position of the head was not noticed.

3. It is assumed that the reaction of the labyrinth was due, in the first mentioned group, to an endolymph flow, whereas in the second group the calorization had finally acted on the end-organ itself.

4. The labyrinthine reflex arc reacts not only to caloric stimuli, but also to constant galvanization, with a tendency toward periodic fluctuations of its excitability.

12. Brody, B. S., and Dusser de Barenne, J. G.: Effect of Hyperventilation on the Excitability of the Motor Cortex in Cats, Arch. Neurol. & Psychiat. **28**:571 (Sept.) 1932.

EXPERIMENTAL SURGERY OF THE NOSE AND SINUSES

II. GROSS RESULTS FOLLOWING THE REMOVAL OF THE INTERSINOUS SEPTUM AND OF STRIPS OF MUCOUS MEMBRANE FROM THE FRONTAL SINUS OF THE DOG

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The results of operation on the accessory nasal sinuses in general are not entirely satisfactory. This is especially true of operations performed on the ethmoid sinuses. With this in mind, the present series of experiments was carried out, together with some studies on the physiology of the nose and sinuses, in the hope of finding possible causes for failure in operations on the sinuses.

In connection with some attempts to produce acute sinusitis experimentally in dogs, the septum between the frontal sinuses of two dogs was removed. The septum was bitten away cleanly and smoothly with rongeurs, including the lining on both sides. A membranous septum promptly reformed. Thinking that the bare strip of bone left after removal of the septum might be responsible in some way for failure of the artificial opening to remain patent, it was decided to remove strips of varying widths from variously shaped surfaces to determine the result.

In all, twenty-four strips of mucous membrane were removed from one or both sinuses of fifteen dogs. They were observed at subsequent operations, after periods of time varying from one day to thirty-six weeks. Each denuded strip has been observed from one to five times subsequent to the operation of denudation.

TECHNIC

All the operations were done under ether anesthesia, employing aseptic technic. The ether was administered through a tracheal tube.

The operation for exposing the sinuses was simple. The frontal bone was laid bare over both frontal sinuses, working through an incision in the median line, and the bony roofs of both sinuses, including the corresponding mucous membrane, were removed at the first operation by means of chisel, mallet and rongeur. After the first operation, depending on the length of time that had elapsed, the bony defect either was found open under the skin or was closed by a tight, fibrous mem-

From the Section on Ophthalmology, the Mayo Clinic. Work done in the Division of Experimental Surgery and Pathology, the Mayo Foundation.

brane that could be opened readily by means of a scalpel. Removal of the roof was modified in some cases in which it was desired, for some reason, to preserve the mucous membrane on the under side of the roof, as in those dogs in which the intersinous septum was removed submucously. In these instances, small openings were cautiously chiseled close to the septum on each side, exposing the lining membrane. This was then separated from the bone by packing wet cotton between the bone and membrane as delicately as possible. The membrane is extremely fragile.

The strips of mucous membrane to be removed were first outlined by means of incisions made by a small, sharp scalpel. They were then removed by means

Summary of Denudations and Results.

Dog	Side of Sinus Operated On	Position of Strip Denuded (Fig. 2)	Length of Time Each Strip Was Under Observation, Weeks	Times Observed at Operations Subsequent to Denudation	Resulting Scar	Interference with Drainage and Collection of Mucin
1	Right	1	3	1	Flat	No
1	.....	3	16	3	Moderately high ridge	No
1	.....	4	11	2	Flat	Yes
1	Left	5	16	3	High ridge	Yes
2	Right	2	17	2	High ridge	Yes
2	Right	8	4	1	High ridge	Yes
3	Left	4	19	4	High ridge	Yes
3	Left	2	19	4	High ridge	No
3	Left	3	19	4	Flat scar	Yes
4	Left	2	18	3	High ridge*	Yes
4	Left	3	18	3	High ridge*	Yes
4	Left	4	18	3	High ridge*	Yes
5	Right	4	13	1	High ridge	No
6	Right	6	8	1	High ridge	Yes
7	Right	6	5	1	Flat scar	No
7	Left	6	12	2	High ridge	No
8	Right	7	16	3	High ridge	Yes
9	Left	7	36	5	Complete diaphragm	Yes
10	Right	7	3	1	Very high ridge	
11	Left	8	16	2	Very high ridge†	Yes
12	Right	9‡	13	1	Low ridge	Yes (at posterior end only)
13	Right	10‖	2	1	Complete diaphragm	Yes
14	Right	10‖	2	1	Diaphragm containing a central perforation	Yes
15	Right	Special#	2	1	Flat scar	No

* These three scars joined together into a very high three-limbed scar that divided the sinus into three chambers, connected only by a comparatively small opening.
† It formed a partition that was two-thirds complete.
‡ Not shown in figure 2. It was on the lateral wall parallel to the direction of drainage.
‖ Not shown in figure 2. It was a circular strip surrounding the ostium.
Not shown in figure 2. It was a circular strip in the posterior part of the sinus.

of a small ethmoid curet or bit of gauze held in the jaws of a small hemostat. Unless an incision was first made, the strips could not be removed cleanly or without making extensive tears in the surrounding membrane. Figure 1 represents a typical frontal sinus with the roof extensively removed. Strips were removed from eight different positions on concave surfaces, as shown in figure 2. The table gives the number and positions of the strips removed from the individual dogs, the length of time of each experiment, and the number of observations made at subsequent operations, together with the results.

RESULTS

The strips within the sinuses, denuded of mucous membrane as described, healed with high, sharp, ridgelike scars in the form of

crescents that spanned the concavities in the walls of the sinus. These scars were well formed after two and three weeks (fig. 3, *A* and *B*). They interfered with drainage when they were so situated, with reference to lines of flow, that mucin could not flow around them.

In all but five of the twenty-four cases, removal of strips resulted in high, sharp scars. In general, the wider the strip denuded, the higher and thicker became the resulting scar. Mucin passed over only two or three of these scars, as indicated by the behavior of india ink which was placed behind the scars, and by the fact that mucin collected there. The five strips which did not form very high scars were all short, and the resulting scars were flat or moderately prominent.

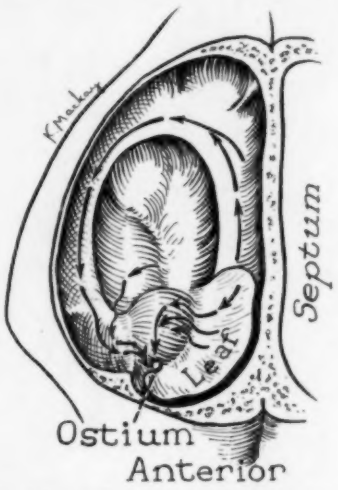

Fig. 1.—Right frontal sinus of a dog, sectioned approximately through the middle, in a plane parallel with that of the frontal bone. The arrows indicate the direction of the flow of mucin moved by ciliary action.

Fifteen of the twenty-four scars were so placed with reference to the lines of drainage that pools of mucin collected behind them (fig. 2). Reference to the arrows in figure 1, or to a previous article in which the details of drainage of this sinus are given, will make clear how this occurs.[1]

One long strip was removed from the lateral wall, parallel with the lines of drainage (not shown in the figures). The resulting scar formed a tunnel that was filled with mucin.

After observing the nature of scars formed following removal of strips of mucous membrane, it was decided to remove some more inter-

1. Hilding, Anderson: The Physiology of Drainage of Nasal Mucus: III. Experimental Work on the Accessory Sinuses, Am. J. Physiol. **100**:664 (May) 1932.

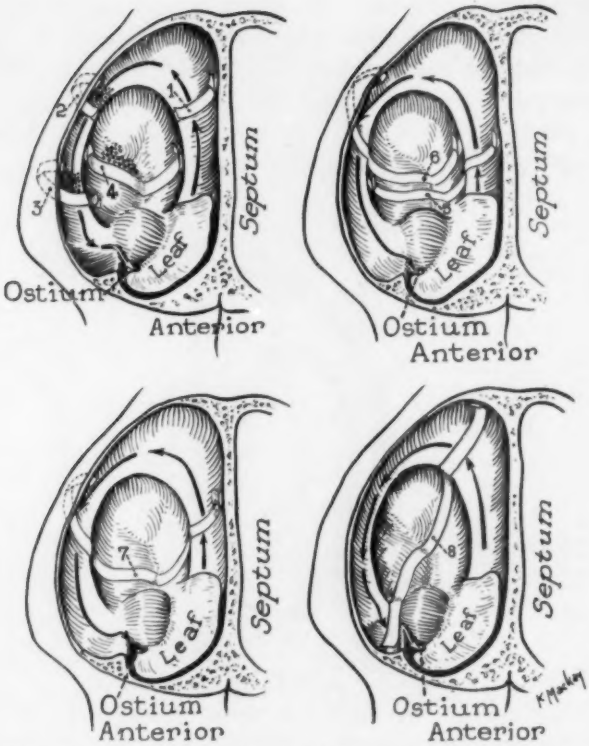

Fig. 2.—Situation of the strips denuded of mucous membrane. A scar in position *1*, does not act as a dam because the mucin flows around the lower end, drawn by the cilia in the deep recess of the sinus. Scars in positions *2* and *3*, on the other hand, are likely to act as dams, and to cause the mucin to pile up behind them, because, first, they are across the line of flow, and second, on the lateral wall there is a tendency for the lines of drainage of mucin to climb upward. The scars meet the operative bony defect at the upper ends, and the mucin cannot flow around; hence, mucin piles up near the upper end. Only one of six strips from these two positions failed to cause collections of mucin. Four strips were removed from position *4*. Two of these resulted in stasis of mucin and two did not. In the latter two, the current of drainage passed around the lateral ends of the scars. One scar each in positions *5* and *6* caused collection of mucin, and two others in position *6* failed to do so. In both these cases the lateral ends of the scars were flat, and drainage occurred directly over them, by means of traction on the overlying film of mucin. Scars in position *7* invariably had collections of mucin at the lateral end; in one dog (dog 9), in which two separate cavities were formed by a complete diaphragm, the posterior cavity was full of mucin. Scars in position *8* also caused much stasis of mucin.

sinous septums in such a way that enough mucous membrane would remain on both sides to cover the bare bone at the base of the septum. It was hoped that by bringing the mucous membrane from the two sinuses in apposition, the formation of a high scar might be prevented. The mucous membrane was therefore gently separated from the septum on both sides, and was conserved while the septum was removed submucously. The membranes were then trimmed with iris scissors, leaving enough to cover the bare bone left by the resection. Three openings made in this way remained patent practically as opened.

To test once more the result of leaving a denuded strip, another septum was removed completely, together with a very narrow strip on each side of the base (fig. 4). This septum reformed promptly as a strong membranous diaphragm.

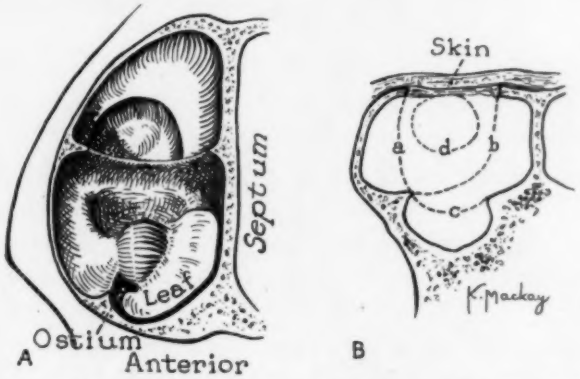

Fig. 3.—*A*, typical appearance of the scars formed after denudation of strips of mucous membrane. This scar, which tended to form a diaphragm, resulted from denudation of a very narrow strip in position *7* (fig. 2) and was eventually considerably higher than shown. The sinus was sectioned in a plane parallel with that of the frontal bone. *B*, diagrammatic representation of a section through the frontal sinus, cut at right angles to the plane of the frontal bone. The dotted lines indicate the outlines taken by the various ridgelike scars. Scars resulting from denuded strips in positions *1*, *2* and *3*, figure 2, formed crescents that reached from the edge of the bony defect in the roof made at the first operation to the edge of the normal horizontal ridge running around the inside of the sinus, as indicated by dotted line *a*. A denuded strip in position *4*, figure 2, healed with a crescent spanning the deep recess, in the plane of dotted line *c*. A scar in position *5*, figure 2, spanned from the bony defect on the mesial side to the horizontal ridge on the opposite side, in the plane of dotted line *b*. Scars in position *6*, figure 2, were similar to those in position *5*, figure 2, except that the scar began at the lateral instead of the mesial margin of the defect in the roof. When a strip was removed across the entire cavity, as in position *7*, figure 2, a circular ridge formed, which grew until it sometimes formed a complete, imperforate diaphragm that divided the sinus into two cavities. Sometimes the diaphragm was incomplete; that is, it had an opening through the center as indicated by dotted line *d*. Two strips taken from position *8*, figure 2, also resulted in diaphragms, one of which was almost complete.

Three other submucous operations were performed. Again all openings remained patent. The opening through one decreased by a third on account of a thin ridgelike scar. Apparently the flaps of mucous membrane had not been well approximated at the time of operation.

The intersinous septum was thus removed from nine dogs in all. The opening remained patent, practically as made, in six in which sufficient membrane was conserved amply to cover the base (dogs 3, 4, 7, 10, 13 and 14). In two from which the septum was neatly removed, including the mucous membrane on both sides (dogs 6 and 16), and in one in which it was removed and a narrow strip of membrane was taken from each side of the base (dog 17), a septum of scar tissue promptly reformed, closing the opening completely.

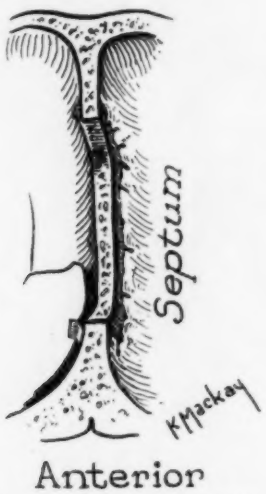

Fig. 4.—Diagrammatic representation of resection of the intersinous septum together with a narrow strip of mucous membrane on each side.

In a third series consisting of seven sinuses of five dogs, an opening was made into one ethmoid sinus, working through the frontal sinus. One of the ethmoid sinuses commonly bulges into the anterior part of the frontal sinus just posterior to the ostium. The wall is usually very thin. Two different technics were used in making the opening. In four sinuses, the opening was made with a small, sharp scalpel through all the layers of the partition (dogs 5, 15 and 17). In this way the edges of the mucous membrane on both sides were in such close apposition that healing would be easy. In the other three dogs (dogs 6, 11 and 17) the sinuses were opened by means of either a curet or scalpel, and a small strip of mucous membrane, a few millimeters wide, was removed from about the opening. This was done either with the curet or by

means of a bit of gauze held in the jaws of a small hemostat, leaving a narrow rim of bare bone about the opening. All three of the latter openings closed tightly in healing. Of the first four, in which the edges of the mucous membrane met accurately, three of the openings remained patent practically as made. The fourth failed to remain open, apparently because at the time the partition was removed the mucous membrane on the inside of the ethmoid sinus separated from the bone and rolled ahead of the scalpel into the bottom of the cavity, and was not satisfactorily incised.

Still another operation was performed on two dogs. A narrow strip of mucous membrane was removed from around the ostium. A diaphragm formed in both after two weeks. In one, the diaphragm was imperforate and closed the ostium completely. In the other, there was still a small opening through the center of the diaphragm.

CONCLUSIONS

From these experiments, the following conclusions may be drawn, at least as far as the normal frontal sinus of the dog is concerned:

1. High ridges and diaphragms of scar tissue follow the removal of strips of mucous membrane on concave surfaces.

2. These ridges and diaphragms interfere with normal drainage, and cause mucin to collect in pools if they are so placed that the mucin cannot readily slide around them.

3. When a complete ring of mucous membrane is removed from the inside of the sinus in any plane, dividing the mucous membrane remaining into halves, the circular scar that forms in healing may become a complete diaphragm of connective tissue, dividing the sinus into two cavities. In that case, one of the cavities subsequently becomes filled with mucin.

4. Partitions, or septums, between sinuses can be removed, and the resulting opening can be caused to remain patent, as made, if the edges of the mucous membrane on both sides of the partition are made to meet, and no strip of bone is left bare.

5. On the other hand, if at the end of the operation a bare strip of bone encircles the opening, a diaphragm forms in healing that usually closes the operative opening and makes the partition or septum intact once more.

6. The ostium can be closed by removing a circular strip of mucous membrane from around it.

TUBERCULOUS MASTOIDITIS IN INFANTS AND IN CHILDREN

REPORT OF CASES

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LOS ANGELES

Tuberculous infection of the mastoid is of clinical importance, particularly in infants and in children, and a study of the literature reveals that this disease has not received the consideration that has been accorded to other entities. In a discussion of mastoid infection, some mention must be made of the middle ear, as its association with the mastoid is intimate. Turner and Fraser [1] reported that in 1,797 cases of suppuration of the middle ear, of the patients under 15 years of age, 2 per cent were tuberculous; under 5 years, 9 per cent; under 2 years, 27 per cent and under 1 year, 50 per cent. McCart [2] found the greatest incidence between the ages of 2 and 5 years.

According to Coates,[3] mastoiditis in infants, to be distinguished as a separate entity from otitis media, must mean infection of the antral mucosa, of the bony walls of the antrum and of the few surrounding cells that may be present, sufficient to cause local symptoms, which may or may not be linked in diagnosis with this local infection.

Tuberculous mastoiditis is usually secondary, and is found in that stage of the disease in which there is an extension of the infection from the original focus, either through the eustachian tube or by way of the lymphatics or blood stream. It is commonly found with tuberculosis of the lungs, intestines, lymph glands, mucous membranes and other organs of the body. The peculiar anatomy of the eustachian tube is probably responsible for a great many of the infections of the middle ear in the infant, in whom the tube is short, broad and more open than in the older child. Tonsils and adenoids, even if not themselves tuber-

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Thesis submitted to the Faculty of Otolaryngology of the Graduate School of the University of Pennsylvania in partial fulfilment of the requirements for the degree of Master of Medical Science (M.Sc. [Med.]) for graduate work in otolaryngology.

1. Turner, A. Logan, and Fraser, J. S.: 1914 Report of Department of Royal Infirmary, Edinburgh, Laryng., Rhin. & Otol. **30**:209 (June) 1915.

2. McCart, H. W. D.: Tuberculous Disease of the Middle Ear, J. Laryng. & Otol. **40**:456, 1925.

3. Coates, George Morrison: Ann. Otol., Rhin. & Laryng. **36**:913 (Dec.) 1927.

culous, may be a factor in establishing this infection in the middle ear and mastoid.

In bottle-fed infants and in younger children, the infection is more apt to be of the bovine type. This is especially true in the rural districts. Various writers have found the incidence of this type of infection in children under 5 years to be between 50 and 75 per cent. Modern methods used by the dairy industry, which include testing of cows with tuberculin, no doubt have reduced the number of these infections.

Mexican children in this locality seem to be more susceptible than others to tuberculous infections, and histories of contact with persons known to be tuberculous are usually obtained. In many cases in which the tubercle bacillus had not been found, patients have received prolonged treatments of various kinds with disappointing results. A history of pulmonary involvement, which on general and roentgen examination would make one suspect the presence of tuberculous infection either in the lungs or elsewhere, would, of course, suggest that an existing infection of the ear might be of a similar nature.

A majority of these infections in the ear begin painlessly and are not recognized until there is a visible discharge from the external canal. Turner [1] found absence of pain in 92 per cent of cases, and the first sign was discharge from the ear and enlargement of glands in 95 per cent. In case 1 reported in this article there was no complaint of pain, and myringotomy was done with only slight discomfort to the patient. Pain may be a feature when there is a mixed infection at the onset, but this is rare. There may also be a slight rise in temperature and in older children a feeling of discomfort in the ear affected, together with impairment of hearing which, as a rule, is marked, both before and after perforation. Tinnitus likewise may be present.

General malaise and loss of weight can usually be expected. In two of the cases presented, however, there was little in the general appearance suggestive of a tuberculous infection of the mastoid. In a few cases diarrhea, vomiting and dehydration may develop, and the infants may lose weight rapidly.

Both ears are usually involved. If the drum is still intact, it frequently has a dull red appearance, becoming pale gray or spotted, as a rule, before spontaneous perforation occurs. At times, only a pinpoint perforation may be seen; later, numerous small openings may be observed which tend to coalesce and form larger openings. Multiple perforations are considered strong evidence of a tuberculous infection.

The discharge is at first very thin, and as the infection involves the mastoid antrum, it becomes profuse and at times irritating to the skin of the external canal and auricle, with resultant dermatitis which is difficult to treat. Subsequently, the discharge becomes thicker and

more purulent, owing to the presence of other organisms. In cases of long standing, the discharge may be fetid, and small particles of bone are occasionally seen in the external canal. The ear drum is rapidly destroyed, and chronic cases usually show granulations on the remaining portions of the drum and middle ear. Destruction of mucous membrane and necrosis of ossicles and adjacent structures soon follow.

Tuberculous mastoiditis is usually more acute in infants and children than in adults. Regional enlargement of lymph glands, at times with marked cervical adenitis, is seen in practically all cases, and is often characteristically painless. Preauricular glandular enlargement is a fairly constant index to aural tuberculosis. Subperiosteal abscesses, a frequent finding, likewise make a painless appearance. Case 1 presents an odd finding, with a perforation of the tuberculous infection through the posterior wall of the external auditory canal, the drum remaining intact. In many tuberculous mastoids not under observation, a painless mastoid infection is in progress before spontaneous discharge attracts attention to the ear.

Facial paralysis is a common finding, resulting from extension of the necrosis to the facial canal, and when present is usually pathognomonic of tuberculous mastoiditis. McCart,[2] in examining 546 patients with tuberculosis of the middle ear, found facial paralysis in 39 per cent of the children, but none in the adults.

Labyrinthitis occurs frequently but in infants is usually not recognized until late, and even then may be overshadowed by findings of meningitis. In children, tuberculous labyrinthitis has an insidious onset. Vertigo of a mild nature is usually present from the start, and later disturbances of equilibrium may be evident. Varying degrees of deafness may be found, and hearing may diminish until it is absent, owing to extension of the pathologic process to the inner ear.

Primary infection of the mastoid is rarely observed. Three cases were reported by Kleinschmidt and Schurmann[4] in which the first symptom was swelling near the ears. The ears started to discharge from one to five weeks later, and then characteristically, but not infallibly, facial paralysis developed. In these cases no form of treatment was effective. Just how the tubercle bacillus gained access to the tympanic cavity was not determined. In many cases reported as instances of primary infection of the mastoid the condition is undoubtedly secondary. The primary focus probably exists, but is not clinically demonstrable.

The diagnosis clinically is not difficult if the following points are kept in mind: the age; the general health; a painless, insidious onset of

4. Kleinschmidt, H., and Schurmann, P.: Primary Tuberculosis of Middle Ear in Early Infancy, Monatschr. f. Kinderh. **40**:193 (Sept.) 1928.

suppuration of the ear; the duration of the discharge; a history of contact with persons known to be tuberculous, and, in the case of bottle-fed infants, the source of the milk supply. The presence of tuberculosis elsewhere in the body, especially in the lungs, is suggestive. The infection is usually bilateral, and impairment of hearing is marked. Spontaneous rupture of the drum not infrequently occurs late, after definite mastoiditis is established. Multiple perforations of the tympanic membrane and extensive granulations on the drum and in the middle ear are commonly observed. The occurrence of facial paralysis is significant; likewise, adjacent glandular enlargement, particularly in the preauricular area. The finding of the tubercle bacillus in the discharge is the final proof of an accurate diagnosis. A positive tuberculin reaction may be of some help. Roentgenograms are of questionable value in infants. In older children, a roentgenogram may show some cloudiness or evidence of bony destruction in the mastoid process, but sheds no light on the nature of the mastoid infection. Mild labyrinthine symptoms when observed with one or more of the foregoing findings, with little or no elevation of temperature, may signify advanced infection of the mastoid involving the inner ear.

The finding of the tubercle bacillus is not accomplished without considerable difficulty. The number of bacilli may vary, but at the onset they are usually plentiful. In cases in which the discharge is of long standing, repeated smears may be negative. The bacillus is more apt to be found if the external canal is cleansed of the bulk of the discharge and the smear taken directly from the middle ear through the perforation. When several smears do not show the bacillus, inoculation of a guinea-pig may be made. This is a slow, but fairly reliable method. When the bacilli are found in large numbers and with relatively little difficulty, an acute process is usually present. Microscopic examination of the granulations obtained from the middle ear or external canal will usually reveal the tubercle bacillus. Cox and Dwyer [5] described a method of growing the bacillus, using Miller's culture medium, which they report gives satisfactory results, growth taking place in from ten to twenty days, after which subcultures are made.

Tuberculin tests should be made as a routine. A positive reaction does not, however, signify that the ear is necessarily tuberculous. Lobban [6] expressed the belief that the earlier the stage of the disease and the less the degree of infection, the greater the dilution of tuberculin with which a positive reaction can be obtained. As the disease

5. Cox, G. H., and Dwyer, J. G.: Tuberculosis of the Middle Ear, Arch. Otolaryng. **9**:417 (April) 1929.

6. Lobban, J. W.: Tuberculin Reaction in Prognosis of Tuberculosis, Tubercle **12**:19 (Oct.) 1930.

advances and the patient's resistance is lowered, the lesser dilutions are necessary before a positive reaction can be obtained. When a good reaction is obtained by a greater dilution, the patient is improving. I have found this to be true in a study of the tuberculous patients at the Children's Hospital, where as a routine 0.1 mg. of tuberculin is used. If the reaction to this amount is negative, 1 mg. is used. Very weak or acutely ill patients may at times give negative or only faintly positive reactions with even 1 mg. of old tuberculin. A bovine tuberculin reaction is made in all cases of disease of the ear in which there is a possibility of tuberculous infection.

Tuberculous mastoiditis is not as often fatal in children as it is commonly thought to be. However, the prognosis depends largely on the extent of the infection in other parts of the body. When the condition in the ear is part of an active general tuberculous infection, the prognosis is, of course, unfavorable. When the lung is apparently the only other organ affected, the outcome depends to a great extent on the progress of the condition in the chest and the resistance of the patient.

The disease has an acute course at the onset, particularly in infants under 1 year of age. Tubercles undergo central necrosis, and exfoliation of the mucosa lining the middle ear and mastoid antrum results, owing to thrombosis of terminal blood vessels. The tympanic membrane and soft tissues of the middle ear, as a rule, are the first parts involved. Necrosis of the ossicles and walls of the middle ear soon follows, together with breaking down of the mastoid cells. Involvement of the facial canal is common, and when it occurs the prognosis is usually unfavorable. The carotid canal may be eroded in a similar manner. Necrosis of the labyrinth is more frequently found as a complication in tuberculous mastoiditis than in any other form of mastoiditis. Turner [1] found this complication in 22 per cent of cases. Fistulas into the labyrinth are frequent. A tuberculous infection may be localized in one part of the labyrinth for some time and not until later become diffuse. The prognosis, as a rule, is unfavorable when this complication exists. Early labyrinthitis may be amenable to timely surgical measures.

Intracranial complications other than tuberculous meningitis are rare. This is especially true of lateral sinus thrombosis, even though the sinus plate is involved in the suppurative process. Tuberculous meningitis is a frequent finding as a cause of death, particularly in infants, in whom the incidence of miliary tuberculosis is high.

Autopsy often reveals extensive cortical perforations, and at times necrosis of the petrous pyramid or of the entire temporal bone. The antrum and mastoid are usually found to contain caseous material, a

fairly constant finding in tuberculous mastoiditis. At times a thin, foul-smelling discharge is present. Bare areas of bone may be found on denuded portions of the middle ear where the mucous membrane has sloughed away. Microscopic examination of granulations and mucosa of the middle ear shows characteristic round cell infiltration and giant cell formation.

Surgical intervention as a rule is indicated and may not only cure the mastoid infection but have a good effect on the general condition of the patient. Less time can be spent on conservative measures than in uncomplicated tuberculous otitis media; however, in patients not acutely ill some treatment may be given to good advantage before an operation is attempted.

Myringotomy is often necessary. The fact that the drum is still intact does not indicate that a mastoid or antral infection is not present. As will be seen in two of the cases presented, involvement of the mastoid was present early, before the drum was opened.

Local treatment consists of daily cleansing of the external canal with hydrogen dioxide on a swab, followed by the application of boric acid in alcohol drops. Removal of tonsils and adenoids may be of some value, as many of these not only form an actual obstruction, but contain large numbers of tubercle bacilli. In infants, adenoidectomy, either before or after the operation, is usually indicated. If this is done, no anesthetic should be used.

The general resistance of the patient is important, and such matters as rest, diet, cod liver oil, sunshine and ultraviolet irradiation with the mercury vapor arc in quartz should be considered both for the conservative and the postoperative treatment. Early, adequate operation is advisable, although the general condition of the patient, of the lungs in particular, should determine the course to be followed. If the condition of the chest is quiescent and the general resistance is good, operation should not be delayed. When the pulmonary lesion is far advanced, or if the mastoid infection is merely part of a general active tuberculosis, operation is usually of little value.

In infants in whom the ossicles are intact, a simple antrotomy may be sufficient. Cell development, however, is frequently encountered in very young infants, and a more complete operation is indicated. In older infants and children with good mastoid development, the success of the operation will depend largely on whether or not all of the involved tissue has been removed. In most cases a radical operation is necessary. According to Graham,[7] infants react quickly to the introduction of tubercle bacilli, and, if the trouble is not recognized early and a radical

7. Graham, Harrington B.: Taking Stock of the Radical Mastoid Operation, Ann. Otol., Rhin. & Laryng. **40**:268 (March) 1931.

exenteration done, disaster will result. He considers the age of an infant no bar to a radical operation. Whenever it is possible to conserve hearing by a modified radical operation, this should of course be done after the physician has made sure that the ossicles are still intact. Some cases appear so far advanced at operation that complete removal of all pathologic material is not possible or advisable. When labyrinthitis is present the patient should be placed in bed under observation, and when the symptoms have subsided as much as possible, a radical operation, adequate drainage being given to the labyrinth, should be performed. When the symptoms have been mild, there is no need to defer the operation.

In small infants, I use 1 per cent procaine hydrochloride without epinephrine for anesthesia. In older infants, 1 per cent procaine hydrochloride with epinephrine is used, supplemented at times with a small dose of sodium amytal by mouth. Children are usually operated on under light nitrogen monoxide anesthesia.

Postoperative drainage is usually profuse, and the wounds show little tendency to heal. Any existing cervical adenitis is usually aggravated for a few days or even weeks after the operation, and at times may suppurate and break down, requiring drainage or removal. Graham [7] advised surgical removal of such enlarged glands, if present, at the time of operation on the mastoid. Wounds occasionally drain for from two to six months, and granulations readily form, which must be kept down by application of caustic or cautery. Local irradiation with ultraviolet rays from the mercury vapor arc in quartz and general ultraviolet irradiation seem to be of definite value in hastening the healing process, and should be started as soon after operation as the temperature has returned to normal. Sun baths in the open air are likewise beneficial as part of the usual tonic therapy and should be continued as long as the weather permits. General dietetic and hygienic care is, of course, essential after operation. The use of tuberculin as part of the postoperative therapy should be mentioned, but just how much good is done is questionable. Transfusions of whole blood for weak or anemic patients either before or after operation are of definite value in increasing the resistance to the infection.

When the tuberculous process has not been successfully arrested by the operation, extension of the infection to the remainder of the temporal bone and labyrinth usually follows, many cases terminating with meningitis or an exacerbation of an existing tuberculous process elsewhere in the body. In a few cases, notably in infants with miliary tuberculous lesions, mere drainage of the mastoid antrum seems to afford some temporary improvement.

REPORT OF CASES

CASE 1.—D. S., a Mexican girl, 2½ years of age, entered the Children's Hospital with a swelling behind the right auricle and a discharge from the right aural canal of six days' duration. There was a history of the patient's having had a severe cold with cough that persisted for two months. The left ear had been draining for eight months. The onset of the discharge in both ears was painless. Her general health had been good, and she had gained in weight steadily. She had been breast fed for eleven months. There was no history of tuberculous contact.

A swelling above and posterior to the right auricle elevated the auricle from the head and extended into the zygomatic area. No tenderness was elicited. The right aural canal was filled with a thin mucopurulent discharge, which came from a red, granulating and bleeding area, ¼ inch (0.64 cm.) from the drum, protruding into the posterosuperior portion of the canal wall. The drum was not red, but showed a general bulging. A myringotomy was done, it being noted at the time that little or no pain attended the procedure. A small amount of serous discharge was obtained. The left drum was red and full, and two small perforations were noted near the center of the drum, through which drained a thin, mucoid discharge. Large adenoids and chronically infected tonsils were present. A small preauricular swelling was evident on the right side and moderate cervical adenitis on both sides.

Physical examination and roentgenograms of the chest revealed scattered tuberculous lesions in the lungs.

Smears from both ears were negative for tubercle bacilli. Roentgenograms of the mastoids were of little value. Some cloudiness was observed on the right, near the aditus. Good cellular development was evident on both sides. One-tenth milligram of tuberculin gave a four plus reaction in twenty-four hours. Hemoglobin was 62 per cent; red blood cells numbered 4,090,000, and white blood cells, 16,900.

The diagnosis was mastoiditis on the right side and otitis media, probably tuberculous, on the left side.

A simple mastoidectomy under nitrogen monoxide anesthesia was done on the right side. A very narrow mastoid was found with the lateral sinus occupying most of the tip, where a few broken-down cells were removed. The perforation through the bony portion of the external canal was part of a tract leading through a necrotic mass of cells to the aditus, which was filled with thick, caseous material. The dura and lateral sinus were not exposed. A smear of material from the mastoid showed numerous tubercle bacilli, and inoculation of guinea-pigs produced the same result.

The temperature rose to 105.2 F. following operation, but soon returned to normal. The wound drained profusely, and healing was slow. Extensive cervical adenitis developed on the right side, which two weeks later broke down and required drainage. The condition of the chest remained unchanged, but the general resistance was good, and a gain in weight was noticed. Sun baths, ultraviolet radiation and cod liver oil were prescribed, and the wound finally closed, leaving a wet, granulating area. The ear canal still contained a small amount of pus. The adenitis persisted, and some drainage continued in the neck. The patient was discharged during the sixth week, and treatments with ultraviolet irradiation were continued in the clinic for three months. During this time the adenitis was markedly reduced and the drainage in the neck stopped. The ear remained moist, and the wound drained a little each day. From the left ear a small amount drained

intermittently, and local treatments with the mercury vapor arc in quartz were given. Tubercle bacilli were finally found in a smear from the left ear. A roentgenogram showed the left mastoid cells clear, with some cloudiness near the aditus. Tonsils and adenoids were removed, and on section and smear tubercle bacilli were found. Measles developed three days later. On the child's return to the clinic at the end of two weeks, the right mastoid wound was completely healed and the middle ear dry, as seen through a large posterior perforation. The left ear was entirely dry. Two small perforations were still present, and the drum retracted. The patient's general condition was good, and the sun baths were continued. At the time of this report, two months have elapsed with no recurrence of the condition.

In this case, the right ear required mastoidectomy. The infection of the left ear, being more or less localized in the middle ear, remained quiescent, and, as the condition of the chest cleared and the general health improved, removal of the tonsils and adenoids was all that was necessary to arrest the infection. Although tubercle bacilli were not found before operation, the mastoid was clinically tuberculous. A painless onset, mastoiditis with an unusual type of perforation and an eardrum still intact are findings suggestive of tuberculous mastoiditis. Repeated tuberculin tests gave four plus reactions, showing a continued good resistance to the infection. An earlier removal of tonsils and adenoids would, no doubt, have shortened the postoperative course.

Case 2.—E. C., a Mexican girl, 3 months of age, entered the hospital with a swelling behind the left auricle, which was of two months' duration. A discharge from the left external canal had been present for two weeks. The swelling had decreased to some extent after the discharge appeared. The patient had been breast fed until the onset of the present trouble, at which time she was given a plain cow's milk formula. There was a history of tuberculous contact.

Complete facial paralysis was present on the left side, which had not been observed by the mother. Pressure over the postauricular swelling forced pus into the external canal through the middle ear. A fluctuating mass was noted in the neck, just below the mastoid tip, on the left side. The canal of the left ear was swollen and filled with a thick, mucopurulent discharge. The ear drum showed a large central perforation. No ossicles were visible. The right ear drum was retracted and dull gray. It was thought at this time that the right ear was free from infection. Cervical adenitis was present on both sides. The tonsils were large and infected. The chest was apparently clear, and the temperature was normal.

Smears of the discharge from the ear were negative for tubercle bacilli. Red blood cells numbered 2,900,000, and white blood cells, 19,000, 60 per cent of which were small lymphocytes.

Under local anesthesia, an extensive subperiosteal abscess over the mastoid process was opened, and a perforation was found in the cortex, which was quite soft. The entire mastoid structure was involved, and another perforation was found in the tip, extending into the neck, where free pus was found under the sternomastoid muscle. A radical operation was done, although the temporal bone was so extensively involved that the procedure appeared hopeless. The wound was left open and a rubber drain inserted in the neck. A smear taken at operation revealed tubercle bacilli.

The temperature reached 102 F. after operation, returning to normal in three days. There was considerable discharge for the next four weeks, and the wound gradually filled in but continued to drain. The facial paralysis persisted. Roentgen examination of the chest showed scattered miliary lesions throughout, suggestive of tuberculosis. One milligram of tuberculin gave only a faint reaction. During the fifth week the temperature was elevated, from 101 to 102 F. Examination of the chest revealed extensive involvement of the lungs. Two milligrams of tuberculin gave a faint reaction. The child finally died.

Autopsy revealed generalized miliary tuberculosis, involving practically every organ of the body. On the side operated on, additional necrosis was found high up on the squamous portion of the bone and deep in the petrous pyramid. On the right side, a circumscribed necrotic area was found near the mastoid antrum, which was filled with caseous material. The middle ear contained only a small amount of seropurulent exudate, and the drum was still intact. Smears from both sides showed tubercle bacilli.

The insidious onset of the infection was evident in this patient, probably starting as part of the general miliary picture. Examination of the right drum showed that apparently the middle ear was not involved. Autopsy, however, showed not only otitis media, but well advanced mastoiditis. Delayed rupture of the drum was also a feature of the infection of the left ear. Operation, though extensive, was of little value, except to establish drainage. The tuberculin reaction in this instance was of more value in prognosis than in diagnosis.

Case 3.—J. M., a Mexican boy of 20 months, entered the hospital with paralysis of the left arm and leg of four days' duration and a cough that had been present for the past two weeks. Five months previously, several hard glands had appeared in the neck, persisting to the time of admission and accompanied by intermittent fever. The child was breast fed for ten months. There was a history of contact with the father and two other children, all of whom had died of tuberculosis.

The patient was semiconscious and presented a flaccid paralysis of the left arm and leg, ptosis of the right eyelid and rigidity of the neck. Cervical adenopathy was present on both sides. The chest contained numerous coarse râles. The ear drums were red, but not bulging. A roentgenogram of the chest showed miliary lesions, probably tuberculous. One-tenth milligram of tuberculin produced a two plus reaction. The temperature varied between 101 and 102 F.

The results of laboratory examinations were as follows: The hemoglobin was 46 per cent; red blood cells numbered 3,870,000 and white blood cells, 17,400. The urine showed 4 plus acetone and no sugar. The spinal fluid cell count was 88. Smears and cultures were negative for tubercle bacilli.

Examination of the ear, nose and throat forty-eight hours after admission showed injected and bulging ear drums. A bilateral myringotomy was done, and a thick, caseous material clung to the blade of the knife. Smears were made of this, and numerous tubercle bacilli were found, confirming the diagnosis of tuberculous otitis media. On the third day in the hospital, the temperature reached 107.6 F. (rectal), and the patient died.

Autopsy revealed tuberculous meningitis and tuberculosis of the lungs and larger lymphatics. Both mastoids were opened, and several cells were encountered filled with caseous material, a few showing necrosis. A smear of this material also showed tubercle bacilli.

The mastoids in this case were the seat of a part of the general infection, and the condition was not the cause of death. Clinically, the picture was that of tuberculous otitis media; actually, however, a well advanced tuberculous mastoiditis existed on both sides, with the ear drums still intact.

CASE 4.—B. S., an American girl, 10 years of age, entered the hospital with a draining right ear of seven and one-half years' duration. Nine months previously the ear had stopped discharging, and the discharge began again one month prior to admission. At the same time, a small swelling the size of a pea appeared in front of the right auricle. Since then, the patient had lost weight and had had frequent "dizzy" spells, which were associated with nausea and headache and were relieved by lying down. There was no history of tuberculosis in the family.

Examination showed some cervical adenopathy; the chest was clear. Neuro-otologic tests were negative, except for 50 per cent loss of hearing on the affected side. Rest in bed at the hospital relieved the nausea and dizziness. The left tympanic membrane was markedly retracted, but otherwise normal. The right middle ear was filled with extensive polypoid granulations, and no portions of the drum or ossicles were visible. The discharge was scant and very foul-smelling. The tonsils and adenoids had been removed previously.

One-tenth milligram of tuberculin gave a three plus reaction in twenty-four hours. Three smears were negative for tubercle bacilli. Physical examination and roentgen examination of the chest gave negative results. A roentgenogram of each mastoid showed sclerosis, and no cells were visible.

Although not proved to be tuberculous, it was felt that such extensive involvement of the middle ear warranted a radical operation. Very hard, eburnated bone was encountered with sclerosis obliterating practically all cells with the exception of a few necrotic areas along the facial ridge and near the aditus. The middle ear was filled with caseous green material, and necrotic portions of the incus and malleus were removed. A direct smear from the mastoid wound revealed tubercle bacilli.

The temperature was normal on admission and rose to 100.2 F. after operation. The wound drained well, and recovery was uneventful. The patient is still under observation.

This case, although operation was certainly indicated, showed that there was a tendency for the lesion to heal, as evidenced by the sclerosis. Nature is at times fairly successful in walling off the disease to the extent that years may elapse without the appearance of complications, and sometimes they never occur.

CASE 5.—W. F., a Mexican boy, entered the hospital at the age of 1 year, with a history of a discharge from the right ear for eleven months and from the left ear for six months. The onset was painless. At 3 months of age he had a "cold" which lasted four months, and which was associated with enlargement of the cervical glands, which persisted. The child was breast fed for nine months, and had direct contact with an aunt who was tuberculous. The patient had previously been treated in the clinic, with no improvement. The mother was not cooperative in bringing the child for examinations. Repeated smears were negative for tubercle bacilli, but inoculation of a guinea-pig finally demonstrated the organisms. One-

tenth milligram of tuberculin produced a two plus reaction. A diagnosis of tuberculous otitis media and possible mastoiditis was made.

At 3 years of age the patient was admitted to the hospital for removal of tonsils and adenoids. The right ear canal was filled with a thick, foul smelling discharge. The drum was red and full and presented a small central perforation. The left canal was filled with similar discharge, and the drum was apparently destroyed, although a mass of granulation protruding from the middle ear obstructed the view. Physical examination and roentgenograms of the chest revealed active tuberculosis, probably miliary, and it was felt that surgical intervention of any kind was contraindicated. The mother took the child from the hospital against advice. The patient was not seen for two years. He had spent most of this time in a warm, dry climate and had received plenty of sunshine. On his return to the clinic, the mother stated that there had been a progressive gain in weight and disappearance of cough, and that the ears had stopped running four months previously. Roentgen examination of the chest showed numerous small lesions apparently healed. Roentgenograms of the mastoids showed no cells on the right and considerable density in the region of the aditus. The left side showed a much larger area of density, apparently sclerosis. Both canals were dry, and the left drum was retracted, exhibiting a small perforation. The right drum showed a large posterior perforation, but the middle ear was dry.

This case, which at one time presented a poor surgical risk, illustrates how a general increase in resistance may be all that is necessary to effect a cure. In this instance, change of climate, fresh air and sunshine played the important rôles.

SUMMARY

1. Tuberculous mastoiditis is common in infants and children. Its incidence and importance are not generally known or appreciated.

2. Routine examination for tubercle bacilli is advisable for all ears with a chronic discharge.

3. Tuberculous mastoiditis may have all or none of the characteristics of mastoiditis due to other organisms.

4. Early diagnosis, careful study, adequate surgical treatment and appropriate postoperative care offer the best possibility of cure.

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THE GENETIC FACTOR IN OTOSCLEROSIS

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(*Continued from page 170*)

PART II. DETAILED DESCRIPTION OF THE VARIOUS MATINGS AND THEIR PROGENY

This material is derived from the field notes of the field workers, the laboratory findings of otologists and the testimony of correspondents.

The evidence is classified into the following chapters: (1) both parents (of propositus) affected, (2) mother only affected, (3) father only affected and (4) neither parent affected.

In each chapter there are given: new pedigrees in which there are affected children all over 30 years of age; pedigrees from the literature in which all the affected children are over 30 years of age; new pedigrees in which there are affected children, some 30 years of age or less and some older, and pedigrees from the literature in which there are affected children, some 30 years of age or less and some older. In the chapter, "Both Parents Affected," because of its importance and the rarity of the combination, certain additional data, less critical and complete, are published.

Throughout our original series "F. H." means family history. The numbers of the family histories are those of the pedigrees (figs. 1 to 48) shown in part II of this paper. Individuals are referred to by the generation and individual number. Thus, "F. H. 26, *II 6*" means family history (and pedigree) 26, generation II and individual 6.

As in some of the larger pedigrees there are several usable matings, the pedigrees are sometimes referred to more than once. Not all of the matings shown are used, however, as in some of the parents or progeny the diagnosis is too unsatisfactory to lay any stress at all on the mating for a study of the inheritance of otosclerosis.

Pedigrees copied from the literature (figs. 49 to 94) are referred to by the name of the author. Full details of the author's diagnostic data are printed in the description of the figures.

In connection with the description of each of the parents and the fraternity of children there is added the probable gametic constitution

on the hypothesis (no. 10) adopted. For brevity's sake the hypothetic gametic constitution is sometimes stated rather dogmatically. Always the meaning is that, in testing out the hypothesis, the formula given is the one that best meets the conditions shown by the parents and children. Of course, in not a few cases the suggested gametic constitution is only one possibility; other possibilities there may well be, though not expressed.

A. BOTH PARENTS AFFECTED

The material for an answer to the question, What is the incidence of otosclerosis in the children when both parents are affected? consists of the following:

I. New pedigrees (3) in which there are affected children: family histories 12, 17 and 22.

II. New pedigrees (2) in which there are, as yet, no affected children: family histories 8 and 42. All 4 children are under 8 years of age.

III. Pedigrees (5) from the literature, in all of which there are affected children: viz., Hammerschlag (1905, table II), Haike (1927-1928, Sa), Bauer and Stein (1925, fig. 6), Gray (1917, table III), and Gray (1917, table VIII).

IV. New pedigrees (6) in which the diagnosis is not certain, but in which there are affected children, some over 30 years of age: family histories 16, 31, 45, 3, 6 and 24.

V. New pedigrees (1) in which the diagnosis is not certain and in which the children are all under 30 years of age: family history 15.

VI. Pedigrees (2) from the literature, in which the diagnosis is not certain and in which the children are under 30 years of age: Bauer and Stein (1925, fig. 7) and Bauer and Stein (1925, fig. 8).

I. *New Pedigrees with Affected Children.*—PEDIGREE 1 (F. H. 12).—The father (*I 9*), aged 72, first noticed deafness in childhood; he still hears fairly well, especially in the right ear. No tinnitus is present. The teeth decayed easily; false ones were acquired at 60 years. Neither of his brothers is known to be affected. His gametes are probably XA, *Y*a; Xa, *Y*A.

The mother (*I 8*), aged 69, first noticed deafness at 40, after all the children were born. There was no known external cause. The deafness is worse in the right ear. The patient does not hear without an instrument. She has roaring tinnitus. Monthly headaches and dizzy spells occurred between 38 and 43 years of age. None of her four siblings is known to be deaf. Her gametes are probably XA, Xa.

Children (6): (1) *II 4*, male, died at 46 years of age without having become noticeably deaf. (2) *II 6*, male, aged 41, has a slight deafness, which began at about 20 years; it does not prevent him from functioning as a golf instructor. (3) *II 8*, male, aged 36, has severe deafness, the onset of which was at about 24. He has dizzy spells and ringing tinnitus. His teeth decayed easily, and have been replaced by false ones. (4) *II 10*, female, aged 45, has severe deafness, requiring aid to

hearing. This was first noticed at 19; it became worse after each pregnancy. The symptoms include headaches and dizzy spells, with roaring and ringing tinnitus. (5) *II 12*, female, aged 44, shows signs of deafness. (6) *II 14*, female, aged 38, has a progressive hardness of hearing, especially in the left ear; this was first noticed at 19. She does not complain of dizziness, but has loud tinnitus and paracusis. Her teeth decay easily.

Comment.—Of 6 children, 5 are progressively deaf, 1 showing the onset at 44 years. The unaffected son died at 46. His zygotic constitution may have been *XY*, aa.

PEDIGREE 2 (F. H. 17).—The father (*II 9*), who died at 72, had a deafness that was not pronounced; the age of onset is not known. He had rather frequent severe sick headaches and considerable sore throat, and acquired false teeth late in life. By an earlier marriage he had 2 deaf sons (*III 17* and *20*), the only children who grew up. Of 3 brothers (*II 11, 16* and *18*) who matured, all had good hearing. Of 2 sisters who matured, 1 (*II 14*) was deaf. This father's father (*I 5*), at the age of 77, had difficulty in following speakers at a meeting. The gametes of *II 9* were probably XA, *Y*a.

The mother (*II 8*) died at 71. Her deafness was first noticed at the age of 36; she could hear with a horn up to the time of her death. The assigned cause of deafness was a fever. There were symptoms of tinnitus (whistling, humming, roaring) and dizzy spells. False teeth were acquired at 50. The siblings are dead. Two brothers (*II 4* and *6*) had good hearing. Of the other 6 siblings nothing is known. The parents (*I 2* and *3*) had good hearing. The gametes of *II 8* were probably XA, Xa.

Children (7): (1) *III 6*, female, aged 70, is deaf. She can hear on the audiphone and indistinctly on the telephone. The deafness is worse in the right ear. Its onset occurred at about the age of 20, and was thought to be due to a severe and neglected cold. The symptoms include tinnitus and occasional dizzy spells. False teeth were acquired at 40. (2) *III 7*, male, died at 63. His hearing is said to have been normal. (3) *III 9*, female, who died at 61 years, had deafness, first noticed at 21; the ascribed cause was "catarrh." The ears never discharged. The symptoms included tinnitus, headaches and frequent colds. False teeth were acquired at 50. (4) *III 11*, male, aged 64, is deaf; however, he can hear a clear voice close to the ear but not on the telephone. Deafness was first noticed between 30 and 35 years of age. The assigned cause was nasal "catarrh." (?) The symptoms included tinnitus (singing, humming and roaring), occasional headaches and frequent sore throat; there was no discharge from the ear. False teeth were acquired at 58. (5) *III 12*, female, aged 62, is deaf. (6) *III 15*, female, aged 59, is deaf. The deafness was first noticed at about the age of 28, after the birth of her second child. The symptoms include tinnitus (ringing, humming and roaring), frequent headaches and vertigo. She had had very few colds in the last twenty years. False teeth were acquired at 50. She had an enlarged goiter removed at 51. (7) *III 16*, female, is a twin of *III 15*. At the age of 59 she is deaf, though she retains a fair amount of hearing, especially when voices are raised, and she answers the telephone. Deafness was first noticed at the age of 23; it developed first in the left ear and then in the right. The history revealed severe headache at 7 years and severe catarrh between 14 and 15. The symptoms include tinnitus (singing, humming and roaring), frequent headaches since 8 years of age, frequent colds and nonresistant teeth, replaced by false ones at 57.

PEDIGREE 3 (F. H. 22).—The father (*II 6*), aged 60, is hard of hearing, more so in the right than in the left ear. This was first noticed at 15 years of age. The ascribed cause is measles. The accompanying symptoms are tinnitus, occasional

headaches, occasional colds and rapid decay of teeth. There has been no discharge from the ears. Of 3 brothers, 1 (*II 9*) is dead; the others (*II 10* and *12*) are not known to have difficulty in hearing. Of 2 sisters, 1 (*II 7*) has been hard of hearing since an attack of scarlet fever in childhood. The father's father (*I 4*) and mother (*I 3*) are both dead; they are not known to have been deaf.

The mother (*II 5*), aged 50, is deaf, especially in the left ear. Deafness was first noticed at about 30 years of age; no cause is ascribed. She had had further loss of hearing after each pregnancy. Her symptoms include occasional headaches, sore throat and many colds, but no tinnitus. Her teeth do not decay markedly. None of her 7 siblings is known to have been deaf.

Her only child, a daughter (*III 3*), aged 27, has deafness which began with itching in the region of the ear at 14 years of age. The deafness was first noticed in the right ear at 15 and in the left ear at 20 years. The symptoms include tinnitus (pulsating and roaring), occasional headaches and frequent sore throat (tonsillectomy was performed at the age of 17). An abscess occurred in the left ear at 24, and 3 impacted teeth were removed at 20. An attack of diphtheria occurred at 19, in consequence of which the legs were paralyzed for six months. Negative results were obtained with the Rinne test.

II. *New Pedigrees with no Affected Children.*—PEDIGREE 1 (F. H. 8).—The father (*IV 14*), aged 43, is deaf; his hearing is worse on the left side. Deafness was first noticed at about 5 years of age; it has gradually increased since childhood, and now amounts to about 40 per cent. The ears have never discharged. The symptoms include tinnitus (whistling, roaring and rumbling), dizzy spells and 1 or 2 headaches a month. The diagnosis is otosclerosis in the third generation. This patient's father (*III 32*) and 2 of his father's sisters (*III 35* and *36*) were similarly affected.

The mother (*IV 13*), aged 38, has deafness which is worse in the left ear. It was first noticed at 31, after her removal to a high, dry location. The deafness was preceded by a tickling irritation of the nose, relieved by liquid petrolatum spray. No discharge from the ears has been noted. The symptoms include tinnitus (ringing), headaches and dizzy spells monthly and rather rapid decay of the teeth. The diagnosis is otosclerosis in the third generation. The father (*III 30*) was also affected, and all 3 siblings (*IV 9, 10* and *12*) have clear symptoms of otosclerosis.

Children (2): *V 3* and *4*, females, aged 4 and 1 years respectively, have no hearing defect.

PEDIGREE 2 (F. H. 42).—The father (*III 7*), aged 40, has an 80 per cent loss of hearing. The deafness began at 20 and was progressive. His 3 brothers (*III 3, 4* and *6*) began to be deaf at 25, 6 and 20 years respectively. Their father (*II 5*) was likewise deaf.

The mother (*III 8*), aged 34, has about a 60 per cent loss of hearing, associated with "catarrh." It was first definitely recognized at 13 years and was progressive. The grandfathers (*I 4* and *6*) on both sides were deaf.

Children (2): In *IV 5* and *6*, both male, aged 7 and 4½ years respectively, no deafness has been recognized.

III. *Pedigrees from the Literature, All with Affected Children.*—PEDIGREE 1 (fig. 74, Hammerschlag,[32] 1905, table II).—The father (*II 7*) had progressive hardness of hearing. Two (*II 3* and *4*) of his 4 brothers, 1 (*II 1*) of his 2 sisters and their mother (*I 2*) were hard of hearing. His gametes were probably XA, *Y*A.

32. Hammerschlag, V.: Wien. klin. Rundschau **19**:5, 1905.

The mother (*III 2*), the niece of her husband, *II 7*, was hard of hearing like her brother (*III 3*), whose deafness was definitely diagnosed as otosclerosis. His gametes were XA, XA.

Children: Two sons and 5 daughters (*IV 1-7*) were all hard of hearing.

PEDIGREE 2 (fig. 67, Haike,[33] 1927-1928, pedigree Sa).—The father (*IV 36*) had deafness diagnosed as otosclerosis. His gametes were XA, *Y*A.

The mother (*IV 35*) was hard of hearing, as were her father's sister (*III 14*) and her father's father (*II 2*). Her gametes were probably XA, XA.

One child (*V 31*), a daughter, had otosclerosis, which appeared at about 25 years of age, after the birth of her third child.

PEDIGREE 3 (fig. 52, Bauer and Stein,[34] 1925, chart 6).—The father (*I 1*) had deafness diagnosed as otosclerosis. His gametes were probably XA, *Y*a.

The mother's (*I 2*) deafness was diagnosed as otosclerosis. Her gametes were probably XA, Xa.

Children (7): (1) *II 5*, male, at the age of 58 was otosclerotic. (2) *II 6*, male, at the age of 52 had normal hearing. (3) *II 1*, female, at the age of 76 was otosclerotic. (4) *II 2*, female, at the age of 67 was otosclerotic. (5) *II 3*, female, at the age of 64 was otosclerotic. (6) *II 4*, female, at the age of 60 had sound hearing. (7) *II 7*, female, who died at 50, was otosclerotic.

PEDIGREE 4 (fig. 57, Gray,[35] 1917, table III).—The father (*II 3*) had otosclerosis in mild degree, first noticed at about 55 years. One of his 7 siblings was hard of hearing between 50 and 60 years of age. The gametes of *II 3* were probably Xa, *Y*A.

The mother (*II 7*) was otosclerotic. Deafness appeared at about 30, but did not progress markedly. Only 1 of her fraternity of 8 was affected. Her gametes were probably Xa, xA.

Children (6): (1) *III 1*, male, at 37 years of age had never had any defect in hearing. (2-3) *III 3* and *5*, female, at 33 and 30 years of age respectively, were carefully examined by Gray; no defect in the ears was found. (4) *III 2*, female, aged 35, had deafness which was definitely diagnosed by Gray as otosclerosis. (5-6) *III 4* and *6*, male, aged 32 and 28 respectively, were carefully examined, and the diagnosis in both was otosclerosis.

PEDIGREE 5 (fig. 61, Gray,[35] 1917, table VIII).—The father (*II 1*), an only child, had bilateral deafness, to a considerable degree, associated with paracusis. Dulness of hearing came on gradually in early middle life. No information was obtained concerning tinnitus. His gametes were probably XA, *Y*a.

The mother (*II 2*) suffered from bilateral deafness associated with paracusis. The presence of tinnitus was not verified. She had 1 brother (*II 3*) with normal hearing. Her gametes were probably XA, Xa.

Children (8): (1) *III 1*, male, died in childhood, before the age at which otosclerosis is likely to appear. (2) *III 2*, female, at the age of 55, had probable otosclerosis, possibly initiated by an attack of scarlet fever. Deafness was bilateral. Tinnitus and paracusis were marked. (3-5) *III 3, 4* and *5*, female, aged 53 to 47, had normal hearing. (6) *III 6*, male, at the age of 45, probably had otosclerosis. The ascribed cause was a fall from a horse. Deafness was first noticed a few weeks after this fall, which was not serious; no symptoms pointed to direct injury of the labyrinth. Tinnitus and paracusis were present. There was no history

33. Haike, H.: Zum Erbgang der Otosklerose, Arch. f. Rassen- u. Gesellsch.-Biol. **20**:155, 1928.

34. Bauer, J., and Stein, C.: Ztschr. f. d. ges. Anat. **10**:483, 1925.

35. Gray, A. A.: Otosclerosis, London, H. K. Lewis & Co., Ltd., 1917.

of suppurative disease or pain in the ear. (7) *III 7*, female, aged 41, had deafness diagnosed as otosclerosis, but the condition was not marked. Symptoms of tinnitus (singing) were present. The ascribed cause was a long period of seasickness. (8) *III 8*, male, aged 38, had bilateral deafness associated with paracusis but without tinnitus. The condition was attributed to colds in the head, but these were probably only the immediate cause.

IV. *New Pedigrees: Diagnosis Not Certain; Affected Children; Some Children Over Thirty Years of Age.*—PEDIGREE 1 (F. H. 16).—The father (*II 7*) was slightly deaf; the onset was first noticed in 1 ear in middle life. His sister (*II 5*) and also their father (*I 2*) were partially deaf for a long time.

The mother (*II 8*) was slightly deaf in 1 ear for a long time. Her siblings and parents were unknown.

Children (9): (1) *III 3*, female, aged 55, is not affected. (2) *III 5*, female, aged 53, is hard of hearing, especially in the left ear. Other symptoms are tinnitus (roaring) and sick headaches. Deafness was first noticed at 45, and was ascribed to "catarrh." (3) *III 7*, female, at the age of 50, has normal hearing. (4) *III 9*, female, at the age of 48, has normal hearing. (5) *III 11*, female, at the age of 45, is deaf, especially in the right ear. She can hear a voice only through a hearing device. Tinnitus (roaring and buzzing) is present. The nose was operated on twice for removal of growths. Tonsillectomies were performed at 13 and 38 years. The patient has had many colds. Her teeth decayed easily; false ones were acquired at 24. Deafness was first noticed at 2 or 3 years of age. The ascribed cause was a severe discharge from the ears. (6) *III 12*, male, is deaf at the age of 42, especially in the left ear. Tinnitus (humming) is present. He has undergone a tonsillectomy and operations on the nose. He has had many colds, and an attack of rheumatism. The defect was first noticed at 32 years. (7) *III 14*, female, at the age of 40, had normal hearing. (8) *III 16*, female, died at the age of 38. Her hearing is believed to have been normal. (9) *III 17*, male, at the age of 36, is hard of hearing. His deafness is ascribed to war service.

PEDIGREE 2 (F. H. 31).—The father (*II 8*) died at 75 years of age. Slight deafness was present from the forty-fifth year on, increasing with age. Its progress is unknown.

The mother (*II 7*) died at the age of 72. Deafness was present, gradually increasing until she was almost totally deaf at the time of her death. The condition was first noticed between 25 and 30 years of age. Tinnitus was present. False teeth were acquired at 60. Her father (*I 3*) was progressively hard of hearing after 30 years of age. Her father's 2 brothers (*I 2* and *5*) were also hard of hearing. Her sister (*II 5*), at the age of 63, is hard of hearing, with the following symptoms: tinnitus (various head noises), sore throat and many colds. She had false teeth at 60. Deafness was first noticed at 55, the ascribed cause being a severe case of influenza. A brother (*II 10*), at the age of 70, is totally deaf. Hardness of hearing began at 25 years, without obvious cause, and progressed until it became total at 55. He has had tinnitus, frequent sore throat and colds, poor teeth and paracusis.

The only child, a daughter (*III 4*), at the age of 40, had about a 50 per cent loss of hearing. The symptoms were tinnitus, sore throat and many colds. A tonsillectomy was performed without improvement in hearing. Deafness was first noticed at 30, without assignable cause; the trouble has increased rapidly.

PEDIGREE 3 (F. H. 45).—The father (*III 13*) died of heart trouble at 68 years. He had otosclerosis on both sides, as demonstrated by histologic study. A quiescent sclerosed area was noted at the anterior margin of the oval window. One (*III 6*) of his 4 brothers, 2 (*III 1* and *8*) of his 3 sisters, 3 of his mother's brothers

(*II 10, 12* and *20*) and 2 of his mother's sisters (*II 14* and *16*) were hard of hearing. His gametes were probably XA, *Y*a.

The mother (*III 14*), aged 62, is hard of hearing. Tinnitus was present at or before the onset of deafness, which was first noticed definitely at about 50 years; it was possibly present at 30. Pregnancy had no effect on the hearing. The ears discharged when she was a child. The Rinne test gave negative results. Her mother (*II 8*) was hard of hearing. Her 4 siblings (*III 15-17*) were normal.

Children: (There were 2 miscarriages.) (1) *IV 13,* male, aged 41, has good hearing. (2) *III 14,* male, at the age of 32, shows what is probably beginning otosclerosis. He has tinnitus and poor teeth, but no earache or discharge from the ears. The Rinne test gave negative results. Bone conduction is increased on both sides. The first symptoms were noted two years ago. The condition has not progressed markedly. The diagnosis was made on the basis of a clinical examination. (3) *IV 15,* female, aged 24, has normal hearing.

Pedigree 4 (F. H. 3).—The father (*II 4*) became deaf at 40. His deafness was not severe. His 4 sisters (*II 5, 7, 9* and *10*) had normal hearing, but 1 of them (*II 10*) had a deaf child (*III 6*) by a man whose 2 sisters were deaf.

The mother (*II 3*) became deaf at 70, the condition progressing to extreme deafness; it was probably not otosclerosis. She had 2 brothers who were not deaf, but 2 aunts who became deaf in middle life.

Children: (1) *III 1,* male, was deaf at 60, but not totally. (2) *III 2,* female, at the age of 50, has a 70 per cent loss of hearing in one ear and a 55 per cent loss in the other. The deafness has progressed after each pregnancy. It was first noticed at 20 years. Both of her sons had mastoiditis.

Pedigree 5 (F. H. 6).—The father (*III 34*) is deaf like his father (*II 20*).

The mother (*III 33*), aged 58, is hard of hearing. The deafness is worse in the left ear. Tinnitus (singing or ringing) is present. She formerly had headaches and much sore throat. False teeth were acquired at 54 years of age. Deafness was first noticed at 54 years following influenza; it is probably not otosclerosis. She has 2 sisters, of whom 1 (*III 32*), aged 54, has defective hearing; it was noticed at 18 and increased after each pregnancy. The other sister (*III 37*), at the age of 45, has deafness which was first noticed at 25 and increased after each pregnancy. Tinnitus (ringing) is present. The teeth decay easily. Two other siblings died early.

Children (2): (1) *IV 30,* female, aged 34, has deafness in the left ear. With the right ear she hears normally. The symptoms are tinnitus (roaring), headaches, vertigo at the age of 27 and sinus infection. She has frequent sore throat and is very susceptible to colds. The teeth are nonresistant. The deafness was first noticed at 27 years, the assigned cause being adenoids and catarrh. (2) Concerning *IV 31,* female, no information was obtained.

Pedigree 6 (F. H. 24).—The father (*II 15*), aged 60, is slightly deaf and very inattentive. His 4 siblings are not deaf. Their mother (*I 16*) was slightly deaf in the left ear at 85 years of age.

The mother (*II 14*), aged 61, has been deaf in the right ear for twenty years or more; the deafness has been progressive. Her brother (*II 12*) was unaffected. Their mother (*I 11*) and father (*I 12*) were both a trifle deaf in old age.

Children (2): (1) *III 2,* female, aged 34, is deaf. She had tinnitus (pulsating) for one year, at the age of 30. Her tonsils were cauterized in 1911 and operated on in 1922. She has colds each winter. The middle turbinate was reduced in 1912. The teeth are in fair condition. The onset of deafness occurred in the right ear ten years ago; in the left ear, thirteen years ago. The ascribed causes were tonsillitis and diphtheria. The Schwabach test was prolonged. The

loss was greater for low than for high tones. The condition was diagnosed as otosclerosis by Dr. Shambaugh. (*2*) *III 3*, male, hears normally.

V. *New Pedigrees: Diagnosis Not Certain; Children All Under Thirty Years of Age.*—PEDIGREE 1 (F. H. 15).—The father (*IV 6*), aged 40, shows deafness which was first noticed on the right side at 32 years and on the left side at 38, becoming rapidly worse on the left. The Rinne test was negative. The eustachian tubes were patent. Inflations gave no relief. The loss in both ears is greatest around 4,000 double vibrations. Nevertheless, it seems probable that this is a case of otosclerosis. Two brothers (*IV 2* and *9*), aged 45 and 38, are hard of hearing. Four sisters (*IV 5, 11, 12* and *14*), from 40 to 25 years of age, are all hard of hearing; 2 (*II 5* and *14*) have tinnitus, with a tendency to colds. The twins (*IV 11* and *12*) are hard of hearing; no further details were secured.

The mother (*IV 7*) shows progressive deafness, first noticed at the age of 20 years. This increased, especially after the birth of the third child. Few headaches, no dizziness and variable tinnitus occur. No symptoms of the nose and throat were mentioned. Colds are uncommon. Two molars have been extracted.

Children (4): (1) *V 4*, male, at 16 years of age, has to sit close to the stage at the theater to hear. The symptoms are vertigo with colds, occasional headaches and chronic pharyngitis. A tonsillectomy was performed at 5 years. No tinnitus is present. Deafness was first noticed in the right ear at 15 years, following pneumonia. The patient was seen by Dr. Harris. Loss of hearing has occurred mostly in the high tones. The condition was diagnosed as otosclerosis. (*2*) *V 5*, female, at the age of 13 years, has a slight deafness not noticed by the patient but involving the loss of 50 units at pitch 80 to 100. The patient has frequent colds. Her teeth are good. No discharge from the ears has occurred. (3) *V 6*, female, aged 10 years, is unaffected. (4) *V 7*, male, aged 4 years, is unaffected.

VI. *Pedigrees from the Literature: Diagnosis Not Certain; Children Under Thirty Years of Age.*—PEDIGREE 1 (fig. 53, Bauer and Stein,[34] 1925, chart 7)—The father (*I 1*) was hard of hearing from an unknown cause.

The mother (*I 2*) was otosclerotic.

Children (6): Of 2 sons, one (*II 5*) was dead at the age of 32 and the other (*II 6*) was living and otosclerotic at 25. Of 4 daughters, 3 (*II 1-3*) were unaffected and 1 (*II 4*), aged 33, had been hard of hearing since 17 years of age.

PEDIGREE 2 (fig. 54, Bauer and Stein,[34] 1925, chart 8).—The father (*I 2*) had labyrinthine deafness.

The mother (*I 1*) was probably otosclerotic.

Children (4): Three sons (*II 1, 2* and *4*), from 43 to 29 years of age, were hard of hearing. A daughter (*II 3*), aged 33, had normal hearing.

B. MOTHER ONLY AFFECTED

The material for an answer to the question, What is the incidence of otosclerosis in the children when only the mother is affected? consists of the following:

I. New pedigrees (11), families with affected children; all of the children are 30 years of age or older: family histories 1, 5, 21, 25, 27, 29, 30, 34, 41, 46 and 48.

II. Pedigrees from the literature (14), families with affected children; all of the children are 30 years of age or older: Hammerschlag (1905, table II), Körner (1905, II), Amadon (1912, p. 927), Amadon

(1912, p. 932), Wojatschek (1925, table IV), Wojatschek (1925, table 7, part 14), Wojatschek (1925, table 7, part 19), Straat (1923), van der Hoeve and de Kleyn (1918, family X), Gray (1917, table XIV), Haike (1927, pedigree Sa), Haike (1928, pedigree Be), Haike (1928, pedigree Be) and (Bronson, 1917, chart 1).

III. New pedigrees (2), families with affected children; the children are both over and under 30 years of age: family histories 37 and 43.

IV. Pedigrees from the literature (5), families with affected children; the children are both over and under 30 years of age: Haike (1927, Pedigree Ne), Gray (1917, table VI), Körner (1905, I), Körner (1905, III) and Haike (1928, pedigree Be).

I. *New Pedigrees: Families with Affected Children; Children All Thirty Years of Age or Older.*—PEDIGREE 1 (F. H. 1).—The father (*II 1*), now dead, was not deaf. His relatives were thought to be free from deafness. His gametes were probably xA, *Y*A.

The mother (*II 2*), who died at the age of 76, had progressive hardness of hearing that began at about 21 years of age. She had headaches, severe tinnitus and sore throat, but no discharge from the ears and no disease of the sinuses or nose. Her teeth were resistant. After 60 years of age, she heard little, even through an instrument. Her mother (*I 2*) was hard of hearing long before her death at 44 years. Her sister (*II 3*) was hard of hearing after about 25 years of age. The deafness was ascribed to scarlet fever. She had constant headaches and tinnitus, but a resistant nose, throat, sinuses and teeth. The mother's gametes were probably XA, xa.

Children (5): (1) *III 1*, male, aged 54, has normal hearing. (2) *III 3*, male, now 50 years old, has had a progressive hardness of hearing since 8 years of age. It did not progress during his sojourn in Cuba between the ages of 25 and 50 years. He has had no headaches, dizzy spells or disease of the sinuses or nose. False teeth were acquired at 49 years of age. Tinnitus occurred before 35 years. (3) *III 5*, female, had a progressive hardness of hearing that was first noticed at 7 years, after mumps; it was formerly accompanied by tinnitus. She was handicapped by the age of 10 years, and is severely deaf at 47, but can use an acousticon. The nose and throat are fairly resistant. The teeth are good. (4) *III 6*, female, has progressive hardness of hearing, first noticed at 18 years. She had a temporary attack of deafness at the age of 30, during which she could hear no conversation. There is no history of headaches, tinnitus or disease of the head or throat. The teeth are only fairly resistant. (5) *III 7*, female, has no defect of hearing. The hearing members of the family are stated to have decidedly better teeth than the others.

Comment.—Otosclerosis is present in 1 of 2 sons and in 2 of 3 daughters. It was to have been expected in 50 per cent of each sex.

PEDIGREE 2 (F. H. 5).—The father (*II 7*), a clergyman, had unaffected hearing. His gametes were probably xa, *Y*A.

The mother (*II 6*), aged 61, formerly a music teacher, first noticed difficulty in hearing at about 20 years of age or earlier. The defect increased rapidly during each pregnancy. She has a loud roaring noise in the head and definite tunes, as though actually heard. No noteworthy infection of the nose and throat has

occurred. False teeth were found necessary at 50 years. One brother (*II 3*), who died at 69, is said to have been somewhat deaf. The gametes of *II 6* are probably xA, XA.

Children (4): (1) *III 5*, female, aged 44, has normal hearing. (2) *III 7*, male, aged 43, has affected hearing in the right ear, ascribed to taking quinine for malaria in the Philippine Islands. There are noises in the head which are not severe. The nose and throat are healthy. False teeth were acquired at 40 years. (3) *III 9*, female, aged 41, has a deafness which began at 15 years; it is worse in the right ear and is ascribed to adenoids. She has occasional headaches but no serious tinnitus. She is not subject to sore throat. She has had sinus infection and operations on the nose and tonsils. The ears have never discharged. The teeth decay easily. She is now very deaf, and hears by the use of a portophone. (4) *III 11*, female, aged 37, first noticed difficulty in hearing at 8 years, affecting mostly the right ear. The loss of hearing was attributed to adenoids. The nose and throat are resistant to infection. The teeth decay easily. Extensive removal of teeth was undergone by the age of 35. The left ear becomes slightly deaf during a cold.

Comment.—Two of the 3 daughters have a progressive deafness; the only son has a deafness ascribed to quinine. The expectation is 50 per cent of each sex affected.

PEDIGREE 3 (F. H. 21).—The father (*II 8*), now dead, was not deaf. His relatives are unknown. His gametes were probably xA, *Y*a.

The mother (*II 7*), aged 77, has a hardness of hearing which was first noticed after her marriage, at 19 years. It is accompanied by constant tinnitus. Her nose and throat are healthy. Her sister (*II 10*), aged 74, has a slowly progressive deafness, which started at the age of 10; severe vertigo is present. The nose and throat are healthy. Another sister (*II 14*), now dead, is said to have been deaf. The gametes of *II 7* are probably XA, xa.

Children (4): (1) *III 25*, female, aged 46, has no deafness. By a normal consort she has 2 sons and 1 daughter. The daughter (*IV 21*), at 17 years, may be showing the onset of deafness. Her right ear drum is said to have been punctured by a fall at 13 years. (2) *III 27*, female, aged 58, is said to be deaf. (3) *III 28*, female, now dead, is believed to have had normal hearing. (4) *III 30*, male, not seen, is said to have normal hearing.

PEDIGREE 4 (F. H. 25).—The father (*III 5*) is believed to have had normal hearing. His gametes were probably xa, *Y*a.

The mother (*III 4*), aged 59, has been progressively deaf since 24 years of age. She has tinnitus, sore throat and liability to colds. The teeth are unresistant; false ones were acquired at 50 years of age. One brother (*III 3*) is not deaf. One sister (*III 1*), aged 65, is slightly deaf. The gametes of *III 4* are probably XA, xa.

Children (3): These include a pair of twins, of whom one (*IV 7*) died in infancy and the other (*IV 8*) is normal. One daughter (*IV 10*) showed deafness during her first pregnancy at 24 years of age; increasing deafness was noted after every succeeding pregnancy. She has had dizzy spells after childbirth, much sore throat accompanied by swelling of glands under the jaw bone, liability to colds and unresistant teeth, but no tinnitus.

PEDIGREE 5 (F. H. 27).—The father (*II 6*) is believed not to be deaf. His relatives are unknown. His gametes are probably xa, *Y*a.

The mother (*II 7*) had increasing deafness after 25 years of age until totally deaf at death. She had 2 deaf brothers (*III 3* and *4*). Her gametes were probably Xa, xa.

The only child who grew to maturity (*III 1*) began to grow deaf at 20 years. She was totally deaf at 40 and died at 52.

PEDIGREE 6 (F. H. 29).—(These data were obtained from the Volta Bureau; the family was not seen.) The father (*II 6*) is said to have had normal hearing. His gametes were probably xa, *Y*a.

The mother (*II 5*) became deaf in middle life, the deafness progressing to totality. She had 7 siblings, of whom 1 died young. One of her 2 brothers (*II 2*) had progressive deafness, beginning at 24 years (like his son and grandson) and becoming total. In all of her 4 sisters (*II 7, 9, 11* and *13*) deafness developed which became marked by middle life. Their mother (*I 1*) became deaf at 35 years, and deafness progressed until total. Her 3 brothers (*I 3*) had normal hearing, as did her husband (*I 2*), whose ancestors are believed to have had normal hearing. The gametes of *II 5* were probably XA, xa.

Children (3): (1) *III 8*, male, is unaffected. (2) *III 9*, male, is also unaffected. (3) *III 10*, female, became deaf after her marriage, and is now very hard of hearing.

PEDIGREE 7 (F. H. 30).—The father (*II 14*) has normal hearing. One of his sisters (*II 13*) is said to have been hard of hearing. His gametes are probably Xa, *Y*a.

The mother (*II 15*), who died at 58 years, was deaf (like her father) from an early age. Her gametes were probably XA, Xa.

Children (3): (1) *III 15*, female, has normal hearing, like her 2 children. (2) *III 16*, male, is deaf (diagnosis uncertain). (3) *III 18*, female, aged 30, has a deafness which was first noticed at about 28 years and which progressed gradually. She has severe headaches, with roaring and sizzling noises in the head. There is no history of infections of the nose or throat. The teeth are good. The diagnosis is otosclerosis.

Comment.—One daughter certainly and probably the only son were affected with otosclerosis, repeating the deafness in the mother and in the father's sister.

PEDIGREE 8 (F. H. 34).—The father (*II 13*) is not known to have been deaf; his relatives are unknown. His gametes were probably Xa, *Y*a.

The mother (*II 12*) became deaf between 30 and 40 years of age. Deafness increased after her sixth pregnancy. Of 9 of her siblings, 3 (*II 5, 8* and *9*) are deaf or became deaf before their death. Her father (*I 2*) was deaf. Her gametes were probably XA, xa and Xa, xA.

Children (5): (1) *III 9*, female, now 74 years old, has a deafness which first became noticeable after 50 years of age; it is accompanied by occasional frontal headaches. Her three pregnancies had no effect on her hearing. The condition is probably not otosclerosis. (2) *III 11*, female, now 68 years of age, has a deafness which was first noticed at 44 years; it is progressive, especially in the right ear, and is accompanied by frequent vertigo and occasional ringing tinnitus. The Weber test was referred to the right. The Rinne test gave positive results. There are only 6 teeth remaining. It is doubtful if the condition is otosclerosis. (3) *III 12*, female, aged 57, is totally deaf in the right ear and uses an instrument at the left ear. Deafness began at about 40 years of age, without assigned cause; it has been accompanied by a few slight headaches. Tinnitus has disappeared, but paracusis is persistent. The sinuses are healthy. The patient has frequent colds and sore throat. The teeth are in very good condition. The Rinne test was positive; the lower tones were not heard. The condition is probably otosclerosis. This woman is one of Dr. Shambaugh's patients. (4) *III 13*, male, is said to have good hearing. (5) *III 14*, male, died in the fourth decade, and was deaf at the time of death.

Comment.—Among the 5 children, all the daughters are hard of hearing. There is some doubt if they are otosclerotic. If so, their zygotic formulas are probably XX, Aa or Xx, Aa. Of the 2 brothers, one is not affected (zygotically x*Y*, Aa) while the other was deaf (zygotically probably X*Y*, Aa).

PEDIGREE 9 (F. H. 41).—The father (*II 2*) had normal hearing. His gametes were xA, *Y*a.

The mother (*II 1*) had a deafness that was noticeable in middle life and that progressed in one ear more than in the other. Her father, (*I 2*) is said to have had a progressive deafness. Her gametes were probably XA, xa.

One child, a son (*III 1*), aged 36, has a deafness that was first noticed at 20 years and that has progressed in the right ear. He has tinnitus, which is not severe, and frequent head colds, but no chronic infection of the nose and throat. These data are from the clinic of Dr. Harris.

Comment.—An only child, a son, has progressive deafness like his mother and mother's father.

PEDIGREE 10 (F. H. 46).—The father (*IV 1*), now dead, was not deaf. Little is known about his relatives' hearing.

The mother (*IV 2*) had been deaf as long as her daughter (*V 12*) could remember. She heard better in a noise. One brother (*IV 3*) had good hearing.

Children (8): Of 3 sons, 1 (*V 1*) became hard of hearing in his middle twenties. Of 5 daughters, 1 (*V 11*) is hard of hearing, deafness having begun when "she was grown."

Comment.—On the assumption of any affected sons and daughters, it is expected that half of them will be affected. One of 3 sons is; another died, unaffected, at the age of 30. Actually, only 1 of 5 daughters is affected, which is markedly below expectation. The evidence is fairly good that the other 4 sisters have good hearing.

PEDIGREE 11 (F. H. 48).—The father (*II 4*) is believed not to have been deaf.

The mother (*II 5*), who died at 78 years of age, had a progressive deafness which became extreme before death, and blue sclerotics. The deafness was not increased by pregnancy.

Children (3): (1) *III 2*, male, had normal hearing at death, at the age of 44. (2) *III 4*, female, aged 66, has been hard of hearing since 40 years of age; the deafness has been progressive, but not affected by pregnancy. She has very blue sclerotics. (3) *III 6*, female, aged 57, is not deaf, but has blue sclerotics.

II. *Pedigrees from the Literature: Families with Affected Children; Children All Thirty Years Old or Older.*—PEDIGREE 1 (fig. 74, Hammerschlag,[32] 1905, table II).—The father (*I 1*) had normal hearing. His gametes were probably xA, *Y*a.

In the mother (*I 2*) an infection of the ear appeared after childbirth, and she gradually became hard of hearing. Her gametes were probably XA, xa.

Children (7): Nothing was known about the hearing of 1 son (*II 5*). Of the 4 other sons, 1 (*II 2*) had normal hearing and 3 (*II 3, 4* and *7*) had progressive hardness of hearing. Of 2 daughters, 1 (*II 6*) was normal and 1 (*II 1*) had progressive hardness of hearing.

Comment.—By hypothesis in this case, half of the sons and daughters should show otosclerosis.

PEDIGREE 2 (fig. 80, Körner,[36] 1905, II).—The father had normal hearing, so far as is known. His gametes were probably xA, *Y*a.

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36. Körner, O.: Ztschr. f. Ohrenh. **50**:98, 1905.

The mother (*II 1*) was deaf. Her gametes were probably XA, xa.

Children (9): Of 4 sons, 2 were normal (*III 1* and *7*) and 2 had hardness of hearing (*III 2* and *3*). Of 5 daughters, 2 were normal (*III 4* and *5*) and 3 hard of hearing (*III 6, 8* and *9*).

PEDIGREE 3 (fig. 50, Amadon,[37] 1912, p. 927).—The father (*I 1*) was believed to have had normal hearing. His gametic formula was probably xA, *Y*A.

The mother (*I 2*) was deaf. Her gametic formula was probably XA, Xa.

Children (5): The eldest son died at the age of 84. He was distinctly gouty or rheumatic; he "had suffered severely from rheumatic arthritis." Autopsy showed otosclerosis, with fixation of the stapes but without degeneration of the eighth nerve or contents of the ductus cochlearis. His only brother (*II 2*) and all 3 sisters (*II 3-5*) were very deaf.

PEDIGREE 4 (fig. 51, Amadon,[37] 1912, p. 937).—The father (*I 1*) had normal hearing, so far as is known. His gametes were probably Xa, *Y*a.

The mother (*I 2*) had been very deaf from youth, like her 5 brothers (*I 3-7*). Her gametes were probably XA, Xa.

Children (7): The eldest (*II 1*), a son, was very deaf at the age of 70. One sibling (*II 7*, sex unknown) is said not to have been deaf, but all 5 of the other siblings (*II 2-6*, sex unknown) were deaf like *II 1*.

PEDIGREE 5 (fig. 89, Wojatschek,[38] 1925, table IV-1).—The father (*II 4*) had normal hearing, so far as is known. His gametes were probably xa, *Y*a.

The mother (*II 3*), her 2 sisters (*II 1* and *2*) and her mother (*I 1*) were considered to have otosclerosis. Her gametes were probably Xa, xA.

Children (7): Of the 5 sons, 3 (*III 5-7*) showed otosclerosis; neither of the daughters (*III 1* and *2*) was affected.

PEDIGREE 6 (fig. 91, Wojatschek,[38] 1925, table 7, part 14).—The father presumably had normal hearing. His formula was probably xa, *Y*a.

The mother (*I 1*) had otosclerosis. Her gametic formula was probably XA, xa.

Children (3): Two daughters (*II 1* and *4*) were affected with otosclerosis and 1 (*II 2*) had normal hearing.

PEDIGREE 7 (fig. 94, Wojatschek,[38] 1925, table 7, part 19).—The father (*III 1*) presumably had normal hearing. His gametes were probably xa, *Y*a.

The mother (*III 2*) had otosclerosis and atrophic rhinitis. Her father (*II 1*) was deaf. One of her brothers (*III 4*) and her only sister (*III 3*) showed progressive deafness. Her gametes were probably XA, xa.

Children (2): One son (*IV 2*) had partial deafness. A daughter (*IV 1*) had normal hearing.

PEDIGREE 8 (fig. 86, Straat,[39] 1923).—The father (*I 1*) had normal hearing. His gametes were probably Xa, *Y*a.

The mother (*I 2*) became deaf at 24 years of age, and had blue sclerotics. Her genetic formula was probably XA, xa.

Children (4): Of 3 daughters, 2 (*II 1* and *4*) were normal. One (*II 2*), aged 37, had become hard of hearing between 10 and 14 years of age and was quite deaf. The diagnosis was otosclerosis. She had brittle bones and blue sclerotics. One

37. Amadon, A. M.: A Case of Otosclerosis with Pathology, Laryngoscope **22**:927, 1912.

38. Wojatschek, W.: De l'hérédité de certaines maladies otolaryngologiques, Rev. de laryng. **46**:541, 1925.

39. Straat, H. L.: Blau Skleren, Fragilitas Ossium und Otosklerose, Nederl. tijdschr. v. geneesk. **2**:151, 1923; Zentralbl. f. Hals-, Nasen- u. Ohrenh. **3**:211, 1923.

son (*II 8*), aged 35, had been hard of hearing since 30 years of age. The condition was diagnosed as otosclerosis. He had frequent fractures in his youth and blue sclerotics.

PEDIGREE 9 (fig. 77, van der Hoeve and de Kleyn,[40] 1918, family X).—The father is believed to have had normal hearing. The gametes were xa, *Y*a.

The mother (*I 1*) died at 63 years of age. She had been very deaf since her fourteenth year, and had brittle bones. Her gametes were probably XA, xa.

Children (6): Of 5 daughters, 1 (*II 1*), who died at 46 years, probably had normal hearing, and 1 (*II 3*) had normal hearing at 49. Of the 3 others, all became deaf in youth; all suffered fractures, and had blue sclerotics. The only son (*II 5*), at the age of 60, had a deafness which began at 22 years, accompanied by loud tinnitus. He had suffered 4 fractures and had blue sclerotics.

It may be added that the youngest of the 5 daughters (*II 6*) had 1 son and 6 daughters (*III 7-13*), all but 1 (*III 13*) with progressive deafness and a tendency toward fractures.

PEDIGREE 10 (fig. 64, Gray,[35] 1917, table XIV).—The father (*I 3*) had normal hearing.

The mother (*I 4*) was deaf.

Children (5): All (*II 3-7*), including both sexes, were otosclerotic.

PEDIGREE 11 (fig. 67, Haike,[33] 1927-1928, pedigree Sa).—The father had normal hearing, so far as is known. His gametes were probably xa, *Y*a.

The mother (*II 1*) was hard of hearing, like at least 2 of her brothers (*II 2* and *5*). The condition of hearing in the other 2 brothers is unknown. Her gametes were probably XA, xa.

Children (9): One of 6 sons (*III 7*) began to lose his hearing at 20 years and was extremely deaf by the age of 40, when he could scarcely hear with a hearing apparatus. He died at 75. Histologic examination showed an ankylosed stapes and degenerative changes in the labyrinth on both sides. The other brothers and sisters were normal, but 2 sisters (*III 5* and *9*) and 1 brother (*III 2*) had affected children.

PEDIGREE 12 (fig. 71, Haike,[41] 1928, pedigree Be).—The father (*II 2*) had normal hearing. His brother (*II 15*) had normal hearing, but 1 of his 6 children (*III 30*) was otosclerotic. The father's gametes were probably xA, *Y*a.

The mother (*II 3*) had otosclerosis, like her brother (*II 11*). Her gametes were probably XA, xA.

Children (9): Of 8 sons, 4 (*III 2, 8* and *9*) had normal hearing and 4 (*III 3-6*) were hard of hearing. One daughter (*III 7*) had normal hearing.

PEDIGREE 13 (fig. 71, Haike,[41] 1928, pedigree Be).—The father (*III 8*) was one of the normal brothers of pedigree 12. His gametes were probably xA, *Y*a.

He married a first cousin once removed (*IV 11*), who was deaf like the brother (*IV 13*), the sister (*IV 15*), the mother of *IV 11* (*III 23*), her 2 sisters (*III 24* and *25*) and their mother's brother (*II 11*). The gametes of *IV 11* were probably XA, xA.

Children: Of their 3 sons, 2 (*V 3* and *4*) had otosclerosis, and the only daughter (*V 1*) was otosclerotic. The other son (*V 2*) was normal.

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40. van der Hoeve, J., and de Kleyn, A.: Blaue Sclera, Knochenbrüchigkeit und Schwerhörigkeit, Arch. f. Ophth. **95**:81, 1918.

41. Haike, H.: Zum Erbgang und zur Konstitutionspathologie der Otosklerose Ztschr. f. Hals-, Nasen- u. Ohrenh. **21**:207, 1928.

PEDIGREE 14 (fig. 55, Bronson,[42] 1917, chart 1).—The father was believed to have had normal hearing.

The mother (*II 1*) was very deaf, with brittle bones like 3 (*II 3-5*) of her 5 sisters. The affected 3 also had blue sclerotics. Their father (*I 1*) was moderately deaf.

Children (6): (1) *III 1*, female, who died in infancy, had blue sclerotics. (2) *III 2*, male, and (3) *III 3*, female, died in infancy. (4) *III 4*, female, aged 39, had been growing deaf since 20 years of age. Her deafness was worse during pregnancy. She suffered from tinnitus and paracusis. Her bones fractured easily, and she had blue sclerotics. (5) *III 5*, female, had normal hearing, white sclerotics and no tendency toward fractures. (6) *III 6*, female, aged 35, had been growing progressively deaf since the age of 16 years. The deafness was accompanied by tinnitus. Her bones fractured easily, and she had blue sclerotics.

III. *New Pedigrees: Families with Affected Children; Children Both Over and Under Thirty Years of Age.*—PEDIGREE 1 (F. H. 37).—The father *(II 8)* is believed not to have been deaf; nothing is known about his relatives' hearing. His gametes were probably xa, *Y*a.

The mother *(II 7)*, aged 59, is deaf. The symptoms were first noticed at 30 years and were ascribed to "catarrh." She has had no discharge from the ears or any loss of hearing after pregnancy. Headaches and dizzy spells occur, and there are noises in the head. No infection of the sinuses or sore throat is reported. The teeth are resistant. A brother *(II 5)* was deaf, supposedly from scarlet fever, the condition beginning at about 30 years of age. He died at 56 years of age. Another brother *(II 4)*, who is believed to have had normal hearing at 58 years, had 1 son *(III 1)* who is slightly hard of hearing at 10 years of age. The mother's father *(I 3)* became hard of hearing at about 30 years; the deafness was ascribed to "catarrh." The gametes of *II 7* are probably XA, xa.

Of 6 children, 1 *(III 13)*, a son, is slightly deaf at 29 years of age.

PEDIGREE 2 (F. H. 43).—The father *(II 6)*, aged 66, hears well. All his siblings died young, and there is no history of deafness in his family. His gametes are probably xa, *Y*a.

The mother *(II 5)*, aged 67, has a deafness that progresses after each pregnancy and that has been diagnosed as otosclerosis. Two of her brothers *(II 3* and *4)*, who died some time ago, are believed to have had normal hearing. Her sister, aged 76, has a progressive deafness; the diagnosis is otosclerosis. The mother's gametes are probably XA, xa.

Children (4): (1-3) *III 4, 5* and *6*, daughters from 38 to 26 years of age, are said to have normal hearing. (4) *III 7*, another daughter, has progressive deafness, which began at the age of 18 years without known cause, accompanied by tinnitus and paracusis. It has been diagnosed as otosclerosis. Her gametic formula is perhaps XA, xa.

IV. *Pedigrees from the Literature: Families with Affected Children; Children Both Under and Over Thirty Years of Age.*—PEDIGREE 1 (fig. 66, Haike,[33] 1927-1928, pedigree Ne).—The father *(II 4)*, at the age of 80, suffered from numerous diseases but not from deafness. His gametes were probably xa, *Y*a.

The mother *(II 2)* was hard of hearing. Deafness first appeared at about 30 years of age, and progressed almost to totality. She died at 72 of a cancer.

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42. Bronson, E.: Fragilitas Ossium and Its Association with Blue Sclerotics and Otosclerosis, Edinburgh M. J. **18**:240, 1917.

Her mother *(I 2)* and her mother's sister *(I 1)* had a similar progressive deafness. Her gametes were probably XA, xa.

Children (2): (1) *III 1,* female, died at 22 of tuberculosis. Her hearing was probably normal. (2) *III 2,* female, aged 40 years, had suffered from hardness of hearing on the left side since the age of 30; she could not distinguish between different instruments in an orchestra. The deafness was progressive and was diagnosed (by Haike) as otosclerosis with involvement of the labyrinth.

PEDIGREE 2 (fig. 59, Gray,[35] 1917, table VI).—The father *(I 1)* was not deaf. No deafness was known among his relatives. His gametes were xa, *Y*a

The mother *(I 2)* had a condition diagnosed as a chronic dry catarrh, a term which at that time included otosclerosis. One sister *(I 4)* was deaf, but no further record could be obtained. Various ancestors were severely affected with deafness. The gametes of *I 2* were probably XA, xa.

Children (6): (1) *II 1,* female, aged 41, began to be deaf at 25; the condition was ascribed to a ride in the wind, followed by otitis media. The deafness had progressed, especially in the left ear, with symptoms typical of otosclerosis. The hearing in the right ear was first noticed as becoming dull at the age of 38. The right ear also had typical symptoms of otosclerosis. (2) *II 2,* male, had no defect of hearing. (3) *II 3,* female, aged 36, had a dulness of hearing which began at the age of 20, accompanied by tinnitus and paracusis. The condition had been diagnosed as otosclerosis. (4) *II 4,* female, had normal hearing. (5) *II 5,* male, aged 25, was affected with deafness accompanied by tinnitus. Dulness of hearing first began at the age of 19; it was twice diagnosed as otosclerosis. (6) *II 6,* male, aged 21, had normal hearing.

Summary.—Of 3 sons, 1 was affected with otosclerosis. Of 3 daughters, 2 were otosclerotic. This approaches the figure of 50 per cent of each sex to be expected from the gametic assumptions made for the parents.

PEDIGREE 3 (fig. 79, Körner,[36] 1905, I).—The father's hearing was normal, so far as is known. His gametes were probably xA, *Y*a.

The mother *(III 6)* had deafness diagnosed as otosclerosis. Her father was deaf. Her gametes were probably xA, Xa.

Children (5): (1) *IV 2,* female, aged 34, was affected with deafness, diagnosed as otosclerosis. (2-3) *IV 3* and *4,* female, aged 30 and 33, had normal hearing. (4) *IV 5,* female, aged 28, was affected with deafness. (5) *IV 6,* male, aged 25, had normal hearing.

PEDIGREE 4 (fig. 81, Körner,[36] 1905, III).—The father is believed not to have been deaf. His gametes were perhaps xa, *Y*a.

The mother *(II 1)* was affected with progressive deafness (otosclerosis), as was her sister *(II 2).* Her gametes were probably XA, xa.

Children (3): (1) *III 1,* male, aged 32, was affected with deafness (2-3) *III 2,* male, and *III 3,* female twins, aged 22, had normal hearing.

PEDIGREE 5 (fig. 71, Haike,[41] 1928, pedigree Be).—The father *(III 16)* is believed to have had normal hearing, but he had 1 brother *(III 18)* and 1 sister *(III 17)* who were hard of hearing. His gametes were probably Xa, *Y*a.

The mother *(III 15)* had been almost completely deaf in the right ear since the birth of her second child at 25 years of age, and the hearing in the left ear was gradually decreasing. Her sister *(III 12)* had had hardness of hearing since the age of 40. The mother's gametes were probably xA, Xa.

Children (3): (1) *IV 3,* female, had had a hardness of hearing, accompanied by tinnitus, since the age of 22. At the age of 38, she found artificial aids to hearing necessary. (2) *IV 4,* male, died at the age of 23. (3) *IV 5,* male, had chronic otitis media.

In addition to the foregoing cases there are certain families in our records—not here analyzed—which contain affected children none of whom are over 30 years old. There are 9 new pedigrees, including 8 male children (25 per cent affected) and 15 female children (53 per cent affected). There are also 8 pedigrees from the literature, including 14 sons (29 per cent affected) and 25 daughters (48 per cent affected). Despite the immaturity of some of the children, we find the tendency toward the proportion of 50 per cent affected, which is generally to be expected when the mother only is affected and is heterozygous for the defect. Finally, our records show fraternities from this mating with no affected children, though all the children are over 30 years of age. This includes 9 new pedigrees with 25 children. There are 16 children in 5 fraternities derived from this mating, taken from the literature, all showing no deafness. We have 3 families in which some of the children are over, and some under, 30 years of age, including 7 sons and 2 daughters, all unaffected; in the literature we find 1 such family with 3 unaffected children. Of children all 30 years of age or less we have 26 new fraternities, comprising 38 sons and 25 daughters, besides 4 children of unknown sex—or 67 altogether —all unaffected. Also, from the literature we glean 17 immature fraternities, including 27 sons and 18 daughters, besides 13 children of unknown sex—or 58 children altogether—all unaffected.

It will be noted that the fraternities comprising unaffected children over 30 years of age are of small size. In the new pedigrees the average size of the fraternity is 2.8; in the pedigrees from the literature, 3.2. This is in contrast with the average size of the fraternity when fraternities all over 30 years of age include affected children. In these cases new pedigrees, including 12 children in 4 fraternities, average 3.0 children in the fraternity. In pedigrees from the literature with affected children there are 81 children in 15 fraternities, or an average of 5.4 children per fraternity.

The foregoing contrast suggests the conclusion that fraternities derived from this mating that have no affected children fall in this group, in part, because of the small number of children. Since it is expected that only a fraction of the fraternity will be affected, the unaffected fraternities may, in part at least, include small fraternities which, by chance, comprise only the unaffected portion of the potentially susceptible fraternities.

The numerous fraternities that include young children are simply not available for statistical analysis, owing to the fact that the incidence of the first recognizable symptoms of otosclerosis is often so late.

C. FATHER ONLY AFFECTED

The material for an answer to the question, What is the incidence of otosclerosis in the children when only the father is affected? consists of the following:

I. New pedigrees (11), families with affected children; the children are all 30 years or over: family histories 2, 8, 8, 8, 14, *26*, 28, *29*, 33, 36 and 38.

II. Pedigrees from the literature (12), families with affected children; the children are all 30 years of age or older: Gray (1917, table XI), Gray (1917, table XIII), Haike (1928, pedigree Be), Hammerschlag (1905, table I), Hammerschlag (1905, table II), Körner (1905, I), Magnus (1876), Undritz (1928, no. 34), Wojatschek (1925, table IV-2), Wojatschek (1925, table 7, part 17), Wojatschek (1925, table 7, part 18) and Wojatschek (1925, table 7, part 19).

III. New pedigrees, families with affected children both over and under 30 years of age: none.

IV. Pedigrees from the literature (4), families with affected children; the children are both over and under 30 years of age: Ruttin (1922, family S), Ruttin (1922, family N), Stobie (1923, fig. 1) and Gray (1917, table XI).

I. *New Pedigrees: Families with Affected Children; Children All Over Thirty Years of Age.*—PEDIGREE 1 (F. H. 2).—The father *(III 5)* was hard of hearing early in life, the deafness progressing until nearly complete; severe tinnitus was present. His father's brother *(II 3)* and his father's mother *(I 1)* were hard of hearing. His gametes were perhaps Xa, *Y*A; XA, *Y*a.

The mother *(III 6)* had normal hearing, as did her brother *(III 7)* and his 2 children (*IV 11* and *12*). Her gametes were perhaps Xa, xa.

Children (5): (1) *IV 2,* female, has a fluctuating hardness of hearing which was first noticed at 33 years, during the last month of gestation. She has frequent headaches, some dizziness and tinnitus, but no infections of the nose and throat. (2) *IV 4,* female, has a hardness of hearing which was first noticed during her twenties, and became severe after her first pregnancy; it has been constant since. There is no history of headaches, tinnitus or infection of the nose and throat. (3) *IV 6,* male, has a hardness of hearing first noticed at about 39 years. He has had some headache and tinnitus, but no infection of the nose or throat. (4-5) *IV 8,* male, and *IV 10,* female, have normal hearing.

Comment.—By hypothesis, one half of the daughters and from one half to one fourth of the sons may be deaf; half of each sex are.

PEDIGREE 2 (F. H. 8).—The father *(III 30)*, aged 64, has had total deafness since he was 50; the condition was first noticed in the left ear at 18 years. He has been subject to frontal headaches and to a ringing, but not disturbing, tinnitus. He is subject to slight attacks of vertigo. There is no history of infection of the sinuses or throat. His teeth are very good. He has been paralyzed on the left side since 30 years of age. His father *(II 29)* and his mother *(II 28)* had normal hearing, but each had 1 sibling who was hard of hearing. His gametes are probably XA, *Y*A.

The mother *(IV 3)* has normal hearing like her 4 siblings, but their father *(III 5)*, father's brother *(III 4)* and father's sister *(III 1)* are hard of hearing; they are the only relatives thus affected in the 3 preceding generations. The gametes are probably Xa, xa.

Children (4): (1) *IV 9*, male, at the age of 30 or 31 years, has defective hearing, the onset of which occurred in the right ear at the age of 17 years and in the left ear at 19. Both ears show a pink inner wall. He is not subject to earaches, but has frequent severe colds. His teeth are very good. (2) *IV 10*, female, aged 33, has a deafness which was first noticed in the right ear at 9 years, and which progressed slowly until after an operation on the ear, when it became rapidly worse. The patient is subject to constant tinnitus and severe headaches. There is a history of infection of the sinuses and tonsils. The tonsils were removed at 25 years, without any improvement in hearing. The patient underwent an appendectomy and an operation on the septum; deafness increased rapidly after each dose of ether. (3) *IV 12*, female, aged 35, has a progressive deafness, first noticed in the right ear at 25 years, after the birth of her first child. Hearing in the right ear is now totally lost, and in the left ear is decreasing gradually. The patient is subject to constant ringing in the right ear, and has frequent colds and sore throat. Her teeth are good. (4) *IV 13*, female, aged 38, has a hardness of hearing, first noticed at 31 years. This is worse on the left side. It began after her removal to a high, dry location. She is subject to headaches and dizzy spells monthly, and to a ringing in the head, and she has occasional sore throat. The teeth decay easily. The condition has been diagnosed as otosclerosis.

Comment.—From the assumed gametic constitution of the parents it is expected that all the daughters and half of the sons will be deaf. The expectation is realized as nearly as possible in this fraternity.

PEDIGREE 3 (F. H. 8).—The father *(III 4)* was hard of hearing throughout middle life, and finally became deaf. Two (*III 1* and *5*) of his 4 siblings are hard of hearing. His parents *(II 3* and *4)* have normal hearing, but his mother had first cousins *(II 7, 8* and *18)* who were hard of hearing. The father's gametes are probably Xa, *Y*A; XA, *Y*a.

The mother *(II 32)* has normal hearing. One of her 7 siblings is hard of hearing, and she has at least 1 nephew, 1 grand-nephew and 3 grand-nieces who are otosclerotic. Her parents had normal hearing. Her gametes are probably xa, Xa.

Children (5): (1) *III 32*, male, has defective hearing. One of his 2 sons *(III 32)* has hardness of hearing which has increased gradually since childhood; his hearing now amounts to about 40 per cent. The condition has been diagnosed as otosclerosis. (2) *III 33*, female, has normal hearing. (3) *III 34*, male, has normal hearing. (4) *III 35*, female, is hard of hearing. (5) *III 36*, female, is hard of hearing.

PEDIGREE 4 (F. H. 8).—The father *(III 32*, the first child in the preceding pedigree) has defective hearing like his father (*III 4*) and 2 of his 4 siblings (*III 1* and *5*). His gametes are probably Xa, *Y*A.

The mother *(III 31)* has normal hearing. Her gametes are probably Xa, xa.

Children (5): (1) A son *(IV 14)*, aged 43, has a hardness of hearing which has been diagnosed as otosclerosis. This was first noticed at 5 years of age, and has always been worse in the left ear. He has tinnitus, headaches once or twice a month and 3 or 4 dizzy spells a year. His hearing is about 60 per cent of normal. The ears have never discharged, and the teeth do not decay

easily. (2-4) Three daughters (*IV 15*) have normal hearing. (5) A son (*IV 16*) has normal hearing.

Comment.—On the assumed gametic constitution of the parents, only half of the sons and none of the daughters should be affected.

PEDIGREE 5 (F. H. 14).—The father *(II 5)* had a hardness of hearing, first noticed at the age of 40; it was ascribed to "catarrh." His gametes were probably XA, *Y*a; Xa, *Y*A.

The mother *(II 6)* apparently had normal hearing. Her gametes were presumably xa, xa.

Children (10): Nine, including 3 sons *(III 15)* and 6 daughters *(III 16)*, were not known to be deaf; their ages at death were not ascertained. The remaining child, a daughter *(III 17)*, who died at 50 years of age, showed a progressive hardness of hearing which began at about 25 years and which was ascribed to "catarrh." The teeth, throat and nose were resistant. Little could be determined concerning the other symptoms shown.

Comment.—By hypothesis, about one half of the daughters and no sons should be affected.

PEDIGREE 6 (F. H. 26).—The father *(II 7)* is known to have been deaf, as was 1 *(II 5)* of his 2 brothers, but no details are known. His gametes were probably XA, *Y*a; Xa, *Y*A.

The mother *(II 8)* is believed to have normal hearing. Her gametes are probably Xa, xa.

Children (7): (1) *III 6*, female, aged 60, has normal hearing. (2) *III 7*, female, aged 58, has a deafness which began with her first pregnancy and increased. (3) *III 9*, male, died in childhood. (4) *III 10*, male, aged 54, has normal hearing. (5) *III 12*, female, is deaf at 52 years. (6) *III 15*, male, is deaf and blind at the age of 50. (7) *III 17*, female, aged 45, has a deafness which was first noticed when she was about 27 years old, before or during her fourth pregnancy. Tinnitus was severe during the first year. The teeth are in poor condition. The Rinne test gave negative results for the left ear and positive results for the right. The patient married twice. By her first husband *(III 16)* she had 2 girls *(IV 17* and *19)* and 1 boy *(IV 21)* who grew up. At their present ages, from 21 to 17, they have normal hearing. By her second marriage, to a normal man *(III 18)*, the patient had 1 daughter *(IV 23)* who, at 14 years, has deafness in the left ear. The defect in hearing was first noticed in the grade school. She has frequent colds, good teeth, infected tonsils and slight vertigo.

Comment.—Since the sons as well as the daughters are affected, the mother must have carried a defective gene in the X-chromosome.

PEDIGREE 7 (F. H. 28).—The father *(III 6)*, who died at 29 years of age, had been forced to give up his law practice because of defective hearing, which was first noticed at 27 years. No cause is ascribed. A similar hardness of hearing was found in his father *(II 4)*, his father's father *(I 2)* and his father's brother *(II 2)*. His gametes were probably Xa, *Y*A.

The mother *(III 7)*, who died at the age of 37, had normal hearing, as did her relatives, so far as is known. Her gametes were probably xA, xa.

The only child, a daughter *(IV 8)*, aged 59, has a gradual progressive hardness of hearing, first noticed at 22 or 23 years. She noticed a loss after the first pregnancy, but some of the loss was regained; much more was lost after the second pregnancy and very little regained. She seldom had tinnitus; it does not bother her at all now. There is no paracusis. Her teeth decayed easily,

and she acquired false ones between 45 and 50 years of age. She had infected tonsils thirty years ago, but they have never been removed. Sore throat or colds are rare, but some nasal discharge occurs.

PEDIGREE 8. (F. H. 29).—The father *(II 2)* had been deaf since about 24 years of age; the condition developed into severe deafness. All of his 5 sisters *(II 5, 7, 9, 11* and *13)* became hard of hearing in middle life. His mother *(I 1)* had a deafness which, starting at the age of 35, progressed to totality. His gametes were probably Xa, *Y*A.

The mother *(II 1)* had normal hearing. Her gametes were probably xa, Xa.

Children (9): (1) A son *(III 1)* had normal hearing. (2) Another son *(III 2)* first noticed deafness at 24 years of age; this has progressed to totality. (3-7) Five daughters *(III 4)* had normal hearing. (8-9) Two sons *(III 5)* had unaffected hearing.

Comment.—By hypothesis none of the daughters, but half of the sons, should be affected. One of the 4 sons is affected.

PEDIGREE 9 (F. H. 33).—The father *(II 6)* has slight deafness, regarded as perhaps the deafness of old age. A sister *(II 1)*, living in Germany, is known to be deaf.

The mother *(II 7)* has normal hearing. One *(II 11)* of her sisters had progressive hardness of hearing, first noticed at 30 years and accompanied by tinnitus. Colds tended to settle in the sinuses.

Children (6): (1) *III 2,* female, a musician, has a hardly noticeable deafness with progressive tinnitus. The Rinne test yielded positive results; bone conduction was shortened on right; the Weber test was referred to the right (the poorer) ear. (2-3) *III 4* and *6,* male, have normal hearing. (4) *III 8,* female, has "catarrhal deafness," first noticed at 18 years, with increasing tinnitus. The mucous membranes of the head are nonresistant. An audiogram made by Dr. Shambaugh shows reduction greatest in conversational tones. The Rinne test gave negative results; bone conduction was prolonged; the Weber test was referred to the left (the better) ear. No discharge from the ears was reported, but paracusis is present. (5) *III 9,* female, has a slight deafness that began with otitis media at 8 years of age; rupture of the right tympanic membrane occurred at 20. Tinnitus is present. The patient has a tendency to head colds. Bone conduction was prolonged; the Rinne test gave positive results on the right. Chronic tubal occlusion is present. (6) *III 10,* female, twin of *III 9,* had a hardness of hearing first noticed at 27 years; tinnitus was constant. She had frontal headaches, but few colds. At 18 years she noticed discomfort from loose cartilages; a roentgenogram showed lack of ossification. The Rinne test gave negative results on the left and positive results on the right. The stapes was found to be fixed.

Comment.—There are varied symptoms in this family complex. If the father is affected with otosclerosis, his gametes should be XA, *Y*a, and the mother's xa, xa. Then all the daughters but no sons should be affected, as is the case.

PEDIGREE 10 (F. H. 36).—The father *(III 11)*, who died at 71, was hard of hearing in 1 ear. The deafness was first noticed when he was a young man. No tinnitus was present. The teeth were resistant, and the tonsils were not removed. His sister *(III 13)*, who died at 81, was hard of hearing from youth. Two other siblings *(III 14* and *15)* had good hearing. Their father *(II 4)*, who died at 86, was decidedly deaf during the latter part of his life. The father's *(III 11)* gametes were probably Xa, *Y*a; Xa, *Y*A.

The mother *(III 10)* is not known to be deaf. Her gametes are probably Xa, xa. She had a sister who was hard of hearing from the age of 56 until death at 66.

Children (3): (1) *IV 10,* male, aged 58, has a progressive deafness first noticed at the age of 22. An abscess of the ear occurred in childhood. He has occasional headaches, and formerly had tinnitus. The nose and throat are fairly resistant. Deafness increased after measles, at the age of 26 (this patient was seen by Dr. Blake of Boston). (2) *IV 11,* male, has had slight deafness in one ear since youth. (3) *IV 13,* female, is hard of hearing; deafness began in middle life.

Three half-siblings of aforementioned children all have a hardness of hearing that began in early life.

Comment.—In the foregoing 4 cases, both the sons and the daughters were affected. The mother's gametes consequently carried the defective gene in the X-chromosome.

PEDIGREE 11 (F. H. 38).—The father (*II 6*), aged 67, seems hard of hearing in one ear. His sister (*II 1*) became hard of hearing at 24 years, with the birth of the first child. His gametes are probably Xa, *Y*A.

The mother (*II 7*) has normal hearing. Her brother (*II 10*) is hard of hearing. Their mother (*I 4*) became hard of hearing at 70 years, as did their mother's brother (*I 5*) at 50 years. The gametes of *II 7* are probably xA, xa.

Children (6): (1-2) Two sons (*III 6*) hear normally. (3-4) Two daughters (*III 7*) hear normally. (5) Another daughter (*III 8*) has a progressive hardness of hearing, first noticed at 21 years, accompanied by headaches, tinnitus and constantly increasing vertigo. She formerly had frequent colds. An operation on the septum was undergone. The femur was broken and did not heal fully for one and a half years. The bones are said to be "soft." (6) One daughter (*III 9*) has normal hearing.

Comment.—If the father's gametes are exclusively Xa, *Y*A, and the mother's xA, xa, about half of the daughters and none of the sons should show otosclerosis. The disease is present in 1 of the 4 daughters and in neither of the sons. There is some evidence of irregular bone formation in *III 8.*

II. *Pedigrees from the Literature: Families with Affected Children; Children All Thirty Years Old or Older.*—PEDIGREE 1 (fig. 62, Gray,[35] 1917, table XI).—The father (*III 2*) had deafness diagnosed by Gray as otosclerosis. His brother (*III 4*) was also otosclerotic.

By his first wife (*III 1*), who was normal, he had 1 otosclerotic daughter (*IV 1*). By his second marriage he had 6 children (*IV 2-7*), 2 of whom were otosclerotic (*IV 3* and *6*) and the others too young to be considered here.

PEDIGREE 2 (fig. 63, Gray,[35] 1917, table XIII).—The father (*III 5*) had deafness diagnosed by Gray as otosclerosis. He had 1 otosclerotic first cousin (*III 7*) and 2 otosclerotic half first cousins (*III 1* and *3*).

The mother (*III 6*) had normal hearing.

Children (11): Of 6 daughters (*IV 1, 3, 4, 8, 10* and *11*), 4 (*IV 1, 8, 10* and *11*) had deafness diagnosed as otosclerosis. Of 4 sons (*IV 2, 5, 7* and *9*), 1 (*IV 5*) had hardness of hearing diagnosed as otosclerosis. There was 1 other child (*IV 6*), of unknown sex.

Comment.—Presumably the father had the gametic formula Xa, *Y*A; XA, *Y*a. The mother's gametes were probably Xa, xa.

PEDIGREE 3 (fig. 71, Haike,[41] 1928, pedigree Be).—The father (*II 11*) was otosclerotic. His gametes were probably XA, *Y*a; Xa, *Y*A.

The mother presumably had normal hearing. Her gametes were xa, Xa.

Children (4): (1) *III 16,* male, had normal hearing. (2) *III 17,* female, was apparently hard of hearing. (3) *III 18,* male, had a hardness of hearing which began at 20 years. (4) *III 19,* male, apparently had normal hearing.

PEDIGREE 4 (fig. 73, Hammerschlag,[32] 1905, table I).—The father (*II 3*) became hard of hearing at the age of 32. He had a partially hard of hearing brother (*II 1*) and a totally deaf sister (*II 2*). Their mother (*I 1*) was moderately hard of hearing. His gametes were probably XA, *Ya*; Xa, *YA*.

The mother is thought to have had normal hearing. Her gametes probably were xA, xa.

Children (4): (1) *III 5*, male, aged 48, had hardness of hearing diagnosed by Hammerschlag as otosclerosis. (2) *III 6*, male, now dead, had normal hearing at the age of 37. (3) *III 7*, female, had normal hearing. (4) *III 8*, female, was hard of hearing.

Comment.—The expectation from the gametic formula is that about half of the sons and half of the daughters will be affected, and this result is found.

PEDIGREE 5 (fig. 74, Hammerschlag,[32] 1905, Table II).—The father (*II 3*) had progressive hardness of hearing like 2 of his brothers (*II 4* and *7*) and 1 of his sisters (*II 1*). Their mother (*I 2*) gradually became hard of hearing after a childbirth. The gametic formula of *II 3* was probably XA, *Ya*; Xa, *YA*.

The mother, as far as is known, had normal hearing.

Children (10): (1) *III 2*, female, was hard of hearing. She was married to her deaf uncle (*II 7*), and had 5 daughters (*IV 1-5*) and 2 sons (*IV 6* and *7*), all hard of hearing. (2) *III 3*, male, the propositus, aged 59, had a hardness of hearing diagnosed by Hammerschlag as otosclerosis. In the course of thirty years, he became extremely hard of hearing. He married a woman of normal hearing, and had 5 children, all with normal hearing. His gametes must have been Xa, *YA*. If his wife's were xa, xa, no defective children were to be expected. (3-10) The remaining 8 children (*III 4*), sex not given, were said to be normal.

PEDIGREE 6 (fig. 79, Körner,[36] 1905, I).—This was published as pedigree III by Hammerschlag.[43] The father (*II 7*) had hardness of hearing believed by Körner to be otosclerosis. He had a sister (*II 8*) who was hard of hearing and probably otosclerotic. His gametes were XA, *Ya*; XA, *YA*.

The mother was believed to have had normal hearing. Her gametes were probably Xa, xa.

Children (5): (1) *III 5*, female, had normal hearing. (2) *III 6*, female, had hardness of hearing diagnosed by Körner as otosclerosis. She was married to a normal man and had 4 daughters, 2 of whom (*IV 2* and *5*) were otosclerotic, and 1 normal son (*IV 6*). Her gametes were probably XA, xa; Xa, xA. (3) *III 7*, male, had hardness of hearing, diagnosed by Körner as otosclerosis. He had 3 children (*IV 7*), all under 18 years of age, with normal hearing. (4) *III 8*, female, had normal hearing. (5) *III 9*, male, had a hardness of hearing diagnosed by Körner as otosclerosis. By a normal wife he had 4 children (*IV 8-11*) under 22 years of age, all with normal hearing.

PEDIGREE 7 (fig. 82, Magnus,[44] 1876).—The father (*I 1*) was hard of hearing. He was married to a normal woman (*I 2*) and had 13 children (*II 1-8*), of whom 7 (*II 1-7*) were deaf. An autopsy on 1 of these, a daughter (*II 1*), showed complete bony ankylosis of the stapes. She had a nephew (*III 1*) already quite deaf in his boyhood years.

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43. Hammerschlag, V.: Zur Aetiologie der Otosklerose, Wien. med. Wchnschr. **58**:563, 1908.

44. Magnus, A.: Ueber Verlauf und Sectionsbefund eines Falles von hochgradiger und eigentümlicher Gehörstörung, Arch. f. Ohrenh. **11**:244, 1876.

PEDIGREE 8 (fig. 87, Undritz,[45] 1928, no. 34).—The father (*II 7*) had deafness diagnosed as otosclerosis. Two sisters (*II 1* and *2*) had "inner ear hardness of hearing," and 2 (*II 3* and *4*) of his 4 brothers and his father (*I 1*) were affected similarly.

The mother (*II 8*) had normal hearing, as had both of her parents (*I 3* and *4*).

Children (4): One son (*III 1*) had a hardness of hearing diagnosed as otosclerosis. The remaining brothers (*III 3* and *4*) and the sister (*III 2*) had normal hearing.

PEDIGREE 9 (fig. 90, Wojatschek,[38] 1925, table IV-2).—The father (*I 1*) was said to show otosclerosis.

The mother (*I 2*) was presumably normal.

Children (6): (1) *II 1*, female, was described as normal. (2-4) *II 2, 3* and *4*, female, had otosclerosis. (5) *II 5*, female, was otosclerotic. (6) *II 6*, male, had normal hearing.

PEDIGREE 10 (fig. 92, Wojatschek,[38] 1925, table 7, part 17).—The father (*III 3*) had incomplete deafness.

The mother (*III 2*) had normal hearing, but her sister (*III 1*) and father's father (*I 1*) were deaf.

Children (2): (1) *IV 1*, male, had deafness diagnosed by the author as otosclerosis following scarlatina. (2) *IV 2*, male, had normal hearing.

PEDIGREE 11 (fig. 93, Wojatschek,[38] 1925, table 7, part 18).—The father (*I 2*) was partially hard of hearing.

The mother (*I 1*) had apparently normal hearing.

Children (3): (1) *II 1*, male, had normal hearing. (2) *II 2*, female, had deafness definitely diagnosed by the author as otosclerosis. (3) *II 3*, female, had an incomplete deafness following scarlatina.

PEDIGREE 12 (fig. 94, Wojatschek,[38] 1925, table 7, part 19).—The father (*II 1*) was deaf.

The mother (*II 2*) had normal hearing but had 1 parent (*I 1*) who was deaf as a result of trauma.

Children (4): (1) *III 2*, female, was observed clinically for deafness diagnosed as otosclerosis. She had an incompletely deaf son. (2) *III 3*, female, was hard of hearing. (3) *III 4*, male, had progressive deafness. (4) *III 5*, male, had normal hearing.

IV. *Pedigrees from the Literature: Families with Affected Children; Children Both Over and Under Thirty Years of Age.*—PEDIGREE 1 (fig. 84, Ruttin,[46] 1922, Family S).—The father (*II 3*), who died of cardiac disease at 56 years, had been hard of hearing since 35 years of age, so that he had to use an ear trumpet. (Huss, who reported the same family, said that he was entirely deaf.) He had blue sclerotics, but it is not known whether he had a tendency toward fractures. He had a brother (*II 1*) and a sister (*II 4*) who were hard of hearing at about 50 years.

The mother (*II 2*) had normal hearing.

Children (7 who matured): (1) *III 1*, female, died at 54 years. Her hearing was already poor at 35. She had blue sclerotics but did not show brittle bones.

45. Undritz, W.: Ueber die Bedeutung der Erbfaktoren bei verschiedenen otorhino-laryngologischen Erkrankungen, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **119**: 270, 1928.

46. Ruttin, E.: Osteopsathyrose und Otosklerose, Ztschr. f. Hals-, Nasen- u. Ohrenh. **3**:263, 1922.

(2) *III 2,* male, died at 53 years. He had been hard of hearing since 22 years of age. He had blue sclerotics and brittle bones. One son (*IV 5*) was deaf. (3) *III 3,* male, aged 52, heard only a loud voice, but did not understand words. He had vertigo but not tinnitus. His deafness was progressive. He had blue sclerotics and brittle bones. (4) *III 4,* female, 38 years of age, had progressive hardness of hearing; the Rinne test gave negative results. She had blue sclerotics and brittle bones. By a normal husband she had 1 son (*IV 9*), who, at 7 years, showed blue sclerotics and brittle bones. (5) *III 5,* aged 37, had somewhat dull hearing. He had blue sclerotics but did not show fragile bones. (6) *III 6,* female, died at 29 years of age. Her hearing was good, and she did not have blue sclerotics or fragile bones. (7) *III 7,* female, died at the age of 15 years. Her hearing was said to have been good.

Comment.—The mother of this fraternity doubtless had a defective gene for otosclerosis in the X-chromosome.

Pedigree 2 (fig. 83, Ruttin,[46] 1922, family N.)—The father (*II 4*), aged 58, had a hardness of hearing which was first noticed at about 21 years. He had blue sclerotics and brittle bones. Two of his sisters (*II 1* and *3*) were similarly hard of hearing. His gametes were probably XA, *Y*a.

The mother (*II 5*) had normal hearing, and showed no brittle bones or blue sclerotics.

Children (*2*): (1) *III 9,* female, 32 years old, had been quite hard of hearing since the age of 16. She had a loud tinnitus and the Rinne test gave negative results. Bone conduction was prolonged. The bones were not brittle. (2) *III 10,* female, 28 years old, had been hard of hearing since 13 years of age. She had blue sclerotics, and had suffered bone fractures. By an apparently normal man she had 2 children *(IV 1* and *2)* whose hearing was good but who had blue sclerotics.

Pedigree 3 (fig. 85, Stobie,[47] 1923, his fig 1).—The father *(II 4),* who died at 63 years of age, became deaf when a young man. He also had blue sclerotics and brittle bones. His father *(I 1),* who died in old age, was reported to have been very deaf and to have had blue sclerotics.

The mother was not reported on. She was presumably normal.

Children (8): (1) *III 5,* female, 49 years of age, was completely deaf. The hardness of hearing began at 13 years. She had a tendency to fall from attacks of giddiness. The enamel of the lower incisors had worn away. She had a deaf daughter (*IV 6*). (2) *III 6,* male, was very deaf, the condition first being noticed in his teens. He was seen by Stobie, who regarded him as otosclerotic. (3) *III 7,* female, was slightly deaf, especially in the right ear. She could hear conversation in a raised voice in her left ear. The condition was diagnosed by Stobie as otosclerosis. Her teeth decayed easily. She had blue sclerotics and brittle bones, a combination which was found in 3 *(IV 10, 13* and *15)* of her children. (4) *III 8,* male, had normal hearing. (5) *III 9,* male, who died at the age of 37, became deaf when a young man. He had blue sclerotics. A daughter *(IV 18)* was deaf and had brittle bones and blue sclerotics. (6) *III 10,* male, had normal hearing. (7) *III 11,* female, aged 29, was hard of hearing. The onset of deafness occurred between 14 and 15 years of age. Her teeth decayed rapidly at the age of 17 years. On clinical examination her deafness was diagnosed as otosclerosis. (8) *III 12,* male, died at 4 years of age.

47. Stobie, W.: The Association of Blue Sclerotics with Brittle Bones and Progressive Deafness, Quart. J. Med. **17**:274, 1923.

PEDIGREE 4 (fig. 62), Gray,[35] 1917, table XI).—The father *(III 2)* suffered from otosclerosis, as did his brother *(III 4)*. Both were apparently seen at the clinic.

The mother *(III 3)* had apparently normal hearing.

Children (6): One *(IV 7)* died young. One daughter *(IV 3)* had otosclerosis and 1 of her siblings (*IV 6*, sex unknown) was also otosclerotic. (The ages of the children were not given.)

D. NEITHER PARENT AFFECTED

A collection was made of pedigrees in which there are 1 or more persons in a fraternity who are affected by otosclerosis and neither the father nor the mother of whom is affected by hardness of hearing. In the simple dominant trait one expects no skipping of a generation in the incidence of the trait, but in the case of a double dominant this is quite possible, on the principle of multiple factors. Thus the mother may have the gametes Xa, xa, and the father the gametes xA, *Y*a. From such gametes will be derived offspring with a zygotic formula Xx, Aa, which will give rise to otosclerotic daughters, or, if the father has the gametic formula xa, *Y*A, some of the sons will have the zygotic constitution *XY*, aA, and will be otosclerotic.

We pass now to a consideration of the cases found, which are as follows:

I. New pedigrees (14), families with affected children; the children are all 30 years of age or older: family histories 3, 6, 8, 9, 10, 13, 19, 20, 23, 32, 39, 42, 44 and 46.

II. Pedigrees from the literature (15), families with affected children; the children are all 30 years of age or older: Albrecht (1922, case 3), Gray (1917, table V), Gray (1917, table VII), Gray (1917, table XIII), Gray (1917, table XIII), Gray (1917, table XIII), Gray (1917, table XV), Haike (1927-1928, Pedigree Sa), Haike (1927-1928, pedigree Sa), Haike (1927-1928, pedigree Sa), Haike (1928, pedigree ma), Haike (1928, pedigree Ri), Haike (1928, pedigree Ri), Haike (1928, pedigree Be) and Undritz (1928, no. 38).

III. New pedigrees (10), families with affected children; the children are both over and under 30 years of age: family histories 4, 7, 11, 18, 35, 40, 44, 45, 46 and 47.

IV. Pedigrees from the literature (7), families with affected children; the children are both over and under 30 years of age: Gray (1917, table II), Haike (1928, pedigree Ma), Haike (1928, pedigree Co), Haike (1928, pedigree Th), Hammerschlag (1906, fig. II), Hammerschlag (1908, pedigree IX) and Kompanejetz (1930, cases 1 and 2).

I. *New Pedigrees: Families with Affected Children; Children all Thirty Years of Age or Older.*—PEDIGREE 1 (F. H. 3).—The father *(I 7)*, now dead, had normal hearing, but 2 of his sisters *(I 3* and *5)* became deaf in middle life. A

third sister *(I 1)*, now dead, had normal hearing. By a normally hearing man *(I 2)* she had a daughter *(II 3)* who was very deaf at her death at 70 years. She married a man *(II 4)* who was hard of hearing at 40 years of age, and both of their children *(III 1* and *2)* were progressively deaf, the daughter *(III 2)* losing some hearing after each pregnancy. The gametes of the father were probably xA, *Y*A.

The mother *(II 10)*, now dead, had good hearing. She was a sister of *II 4*. Thus, the father married a sister of his deaf niece's deaf husband. The mother's gametic constitution was probably Xa, xa.

There is 1 child *(III 6)*, a son, from this mating. He became deaf early in life. He had a hard of hearing daughter *(IV 6)*. The deafness was probably otosclerosis, but precise details are lacking.

Comment.—Two hearing parents with the gametic constitution indicated might have a son, Aa, X*Y*, who would become deaf.

PEDIGREE 2 (F. H. 6).—The father *(III 22)* has normal hearing. By his second marriage, to a deaf woman *(III 32)*, he had a normally hearing son *(IV 26)*, now a student in a medical school. This son possibly may not have reached the age of onset of the defect. The father's gametic constitution is perhaps xa, *Y*A.

The mother *(III 21)* has normal hearing, but 1 of her brothers *(III 15)* is quite deaf. Their mother *(II 8)*, now dead, was also deaf and had 2 sisters *(II 9* and *13)* who were hard of hearing. The gametes of *III 21* are probably Xa, xa.

There is a single child, a son *(IV 24)*, aged 43, who has a progressive deafness which was first noticed at about 7 years of age. The condition was aggravated by frequent swimming and by an attack of typhoid at 19 years of age. There was severe tinnitus from 12 to 35 years and vertigo at 30, following severe exertion. The condition of the right ear was aggravated at 30 years following colds. Inflation does not improve the hearing. No infection of the sinuses or throat and few colds occur. The teeth are good. Hearing is now entirely unserviceable in the left ear, and nearly so in the right, for pitches of from 2,000 to 4,000 double vibrations and above. The deafness is probably otosclerosis, despite the loss of hearing for high notes. The zygotic constitution of *IV 24* is probably X*Y*, aA.

PEDIGREE 3 (F. H. 8).—The father (*II 29*) had normal hearing. One of his sisters *(II 30)* is hard of hearing.

Mother *(II 28)* had normal hearing. She had a brother *(II 18)* and a half brother *(II 8)* who were hard of hearing.

Children (3): (1) *III 28*, female, has normal hearing. (2) *III 29*, female, died in infancy. (3) *III 30*, male, aged 64, has total deafness, the onset of which was at 18 years. The condition has been accompanied by severe frontal headaches, slight vertigo and ringing tinnitus. He has a nasal discharge. His teeth are very good.

Comment.—The father's gametes are probably xa, *Y*A, and the mother's Xa, xa. Some affected and some unaffected children are to be expected.

PEDIGREE 4 (F. H. 9).—The father (*II 4*), now dead, is said to have had normal hearing, like all of his fraternity. His gametes were probably xA, *Y*a.

The mother (*II 3*) had normal hearing, but her mother (*I 3*), now dead, is said to have become totally deaf in her early twenties, before the birth of her first child. The deafness was ascribed to the taking of quinine. At the time of her death, at 65 years, she had a large goiter. Her 3 sisters *(I 4-6)* were

unaffected. She married a man *(I 2)* with normal hearing, whose brother *(I 1)* was deaf in his old age. The mother's gametes are probably Xa, xa.

The only daughter *(III 3)* was seen by Dr. Milles. She has a deafness which is worse in the right ear and which was first noticed at about 28 years of age, following a shock caused by the death of her baby. She had severe frontal headaches when in college, and suffered from vertigo at the onset of the deafness. Tinnitus has been present, but is decreasing. There are frequent colds and much neuritis. Her teeth have been filled repeatedly. She has had 4 children, of whom 1 *(IV 1)*, at the age of 16, shows very slight deafness. The zygotic constitution of *III 3* is probably Xx, aA. This case is from Dr. Dench's clinic, the diagnosis being otosclerosis.

PEDIGREE 5 (F. H. 10).—The father *(II 4)* is believed to have had perfect hearing. His gametes were probably xa, *Y*A.

The mother *(II 3)* had normal hearing. A brother *(II 1)*, aged 67, has a hardness of hearing which is worse in the left ear. This was first noticed at about 40 years of age, and was ascribed to an early earache, accompanied by a discharge. He has headaches much of the time, tinnitus and colds. He acquired false teeth at the age of 45. The mother's gametes are probably Xa, xa.

Children (5♀): (1) *III 2*, male, aged 48, has a hardness of hearing, which is worse in the left ear; it was first noticed at 13 years of age, and is ascribed to a nasal catarrh which followed influenza and for which a nasal operation was performed. The ears have never discharged. He formerly had frequent headaches, but these are less common now. There is tinnitus. He can hear ordinary conversation if it is very clear and if the speakers are not over 2 or 3 feet (60 to 90 cm.) away. He has had considerable repair work done on his teeth. (2) *III 3*, male, aged 55, has a progressive deafness which is worse in the right ear; it was first noticed at about 40 years of age. No immediate factor in the onset of deafness has been determined. The patient is resistant to infection of the nose and throat; he has occasional headaches and suffers severely from neuralgia at times. He can hear ordinary conversation, if very clear, on the left side and not far away. He is married to a normal woman *(III 4)* and has 6 children (*IV 2* and *3*), the eldest of whom is 23 years old; none show deafness. (3) *III 6*, male, aged 44, shows a deafness which was first noticed at about 30 years of age; it was ascribed to catarrh and his vocation of railroad engineer (noise of engine). He suffers much from colds. He is married to a normal woman *(III 5)* and has a son *(IV 4)* of 17 years and a daughter *(IV 5)* of 9 years, both with normal hearing. (4) *III 8*, female, aged 34, is believed to have normal hearing. (5) *III 9*, male, is believed to have normal hearing at the age of 40.

Comment.—Three of the 4 sons are affected, and the daughter is unaffected. By hypothesis, half of the sons and none, or few, of the daughters should be affected.

PEDIGREE 6 (F. H. 13).—The father (*II 3*) is believed to have had normal hearing. Nothing is known of his relatives. His gametes were probably xA, *Y*a.

The mother (*II 4*) died at 56 years of age. She is believed to have been unaffected, as were her 6 siblings (*II 2* and *6-10*), but 1 brother (*II 6*) has a child (*III 6*) who was born deaf. Her gametes were probably Xa, xa.

Children (2): (1) *III 3*, male, aged 38, has normal hearing. (2) *III 4*, female, at 40 years of age, has hardness of hearing, first noticed when she was 3 years old; it is worse on the left side. The ears have never discharged. She has headaches twice a week, dizzy spells nearly every morning, tinnitus and a slight tendency toward sore throat but no infection of the sinuses or nose. Her zygotic constitution is probably Xx, aA.

PEDIGREE 7 (F. H. 19).—The father (*II 9*) is said to have had normal hearing. He died of tuberculosis, as did, at an early age, his 4 brothers (*II 11-14*), all of whom had "a catarrhal deafness." His gametes were probably xA, *Y*A.

The mother (*II 8*) died of tuberculosis at 64 years of age. She is stated to have had normal hearing. Her gametes were probably Xa, xa.

Five children: (1) *III 9*, male, now 62 years old, has hardness of hearing, which began at 45 years without known cause and is worse in the right ear. The ears have never discharged. He has headaches and colds frequently, has had dizzy spells since 30 years of age, and is troubled with tinnitus. No infection of the sinuses or throat is reported. His teeth are poor. He had "rheumatism" at 25 years of age, and possibly has tuberculosis at the present time. (2) *III 10*, female, is stated to have died of tuberculosis at 56 years, and to have had normal hearing. (3) *III 13*, male, at 58 years of age, has normal hearing. (4) *III 14*, female, 56 years old, has about a 60 per cent loss of hearing. This first developed at 30 years without clear cause, and is worse in the left ear. Loss of hearing occurs after each pregnancy. She has frequent sick headaches and dizzy spells, tinnitus, occasional sore throat and frequent colds accompanied by severe pains in the head. She recently acquired false teeth. (5) *III 16*, male, aged 45 years, has a hardness of hearing which first developed at 38 years without inciting cause; it is worse in the left ear. The ears have never discharged. He has weekly headaches and frequent dizzy spells. Tinnitus is present, with a tendency toward colds, but no infections of sinuses or nose. False teeth were acquired at about 38 years. Deafness has progressed, though under electrical treatments it is now somewhat improved.

Comment.—Two of 3 sons and 1 of 2 daughters were affected. The assumed gametic constitution of the parents would result in about half of the sons and half of the daughters being deaf.

PEDIGREE 8 (F. H. 20).—The father (*II 2*) is stated not to have been deaf. His mother (*I 2*) had a sister (*I 4*) who was hard of hearing. By a second marriage to a normally hearing man (*I 3*) his mother had 6 children (*II 3-10*), among whom 1 son (*II 4*) and 1 daughter (*II 3*) had a progressive, early appearing hardness of hearing. The gametic constitution of *II 2* was probably xA, *Y*a.

The mother (*II 1*) had normal hearing. She knew of no deafness among her relatives. Her gametic constitution was probably Xa, xa.

Children (2): (1) *III 1*, female, 35 years of age, has a deafness which was first noticed at 17 years; it is worse in left ear. This is accompanied by slight roaring in the head. No sinus trouble or decay of the teeth is reported. She has occasional sore throat and constant nasal "catarrh." (2) *III 2*, male, had normal hearing, as have his children by a normal wife.

PEDIGREE 9 (F. H. 23).—The father (*II 2*) is said to have had normal hearing, but the other 3 members (*II 3, 4* and *6*) of his fraternity had an early appearing progressive deafness, as did their father (*I 2*). The gametes of *II 2* were xA, *Y*a.

The mother (*II 1*) had normal hearing. Her relatives were unknown. Her gametes were probably Xa, xa.

The only child (*III 1*) was a daughter, now dead, who is stated to have had a progressive deafness, beginning at 19 years of age.

PEDIGREE 10 (F. H. 32).—This shows 2 cases of defective offspring from normal parents. Of the hearing of the older pair of parents (*II 7* and *8*), long since dead, little is definitely known. They had 1 daughter (*III 13*) with deafness diagnosed as otosclerosis and beginning at 29 years of age. There was no vertigo, and the throat was rarely sore. Tinnitus was present.

The father (*III 14*), now dead, had a brother (*III 10*) who died at the age of 68 and who was deaf late in life. A sister (*III 13*) already mentioned, aged 66, has a progressive deafness which was first noticed at the age of 29 following typhoid fever; it is worse in the right ear. She had tinnitus during the first fifteen years of deafness. The father's gametic constitution was probably xa, *Y*A.

The mother (*III 15*) has normal hearing. Her relatives are unknown. Her gametic constitution is probably Xa, xa.

Children (2): (1) *IV 11*, male, has a hardness of hearing that began at 25 years and has been severe since his twenty-eighth year. (2) *IV 13*, female, has normal hearing.

PEDIGREE 11 (F. H. 39).—Father (*II 3*) is stated to have had normal hearing. His sister (*II 1*) and her only child (*III 1*) are said also to have had normal hearing. The father's gametes were probably xA, *Y*a.

The mother (*II 4*) is stated to have had normal hearing. Of her 10 siblings, a sister (*II 15*) is stated to have had hardness of hearing, the onset of which was at about 21 years of age. The mother's gametes were probably Xa, xa.

Children (3): (1-2) Two sons (*III 3* and *4*) have normal hearing. (3) A daughter (*III 6*), aged 43, has a hardness of hearing which was first noticed at 22 years of age (no assigned cause); it is worse in the left ear. The ears have never discharged. She has headaches, occasional dizzy spells when eating and throbbing sounds in the head. She formerly took cold readily, and has had the tonsils removed. No infection of the sinuses or nose is reported. The teeth are good.

PEDIGREE 12 (F. H. 42).—The pedigree contains 2 cases of defective hearing in children of unaffected parents, but since the youngest child of parents *I 1* and *2* died long ago, his case is not considered. In the other case the father is *II 8*. His hearing is normal, but his father (*I 6*) was hard of hearing. The gametes of *II 8* are probably xA, *Y*a.

The mother (*II 7*) has normal hearing, but her father (*I 4*) had defective hearing, thought to have followed scarlet fever. The mother's gametes are probably Xa, xa.

The only child (*III 8*), a daughter, aged 34, has a hardness of hearing (60 per cent loss) first noticed at 13 years; the onset is ascribed to "catarrh."

PEDIGREE 13 (F. H. 44).—The father (*II 14*) has normal hearing. One of his brothers (*II 12*) is hard of hearing. Further details of the history are unknown.

The mother (*II 13*) died at 28 years of age. She had normal hearing and, so far as is known, her parents' hearing was unaffected. There is, however, deafness (otosclerosis) in her first cousins once removed (*III 15* and *21*).

Children: Of 3 sons, 1 is unaffected, 1 died in infancy and 1 (*III 23*) at 35 years, has a difficulty of hearing, first noticed at the age of 14; no tinnitus is present. The tonsils have been removed. His hearing grew worse after an attack of influenza ten years earlier. Paracusis is present.

Comment.—Since both parents have relatives who are hard of hearing, it seems probable that the gametes of the father were xA, *Y*a and xa, *Y*A, and those of the mother, Xa, xa. On this hypothesis, otosclerosis might develop in half of the sons.

PEDIGREE 14 (F. H. 46).—The father (*V 19*) died at 85 years of age. His hearing was normal, like that of his siblings and immediate parents, so far as is known.

The mother (*V 18*) has normal hearing, as (it is stated) her parents had.

Children (7): (1) *VI 17*, male, who died at the age of 67, was not hard of hearing until during the last two or three years of his life, when, in connection with

his final illness, he had a very slight deafness. All the other children have normal hearing excepting 2 daughters; 1 of these (*VI 27*) has progressive deafness, which set in at 25 years of age and is accompanied by paracusis, and the other (*VI 28*) has a deafness ascribed to "scarlet fever at 18 years of age."

II. *Pedigrees from the Literature: Families with Affected Children; Children All Thirty Years of Age or Older.*—Pedigree 1 (fig. 49, Albrecht,[48] 1922, case 3).—The father (*II 2*) had normal hearing, but his father (*I 2*) gradually became hard of hearing after the age of 40.

The mother (*II 1*) had normal hearing. She was not a blood relative of *II 2*.

Children (8): (1) *III 1*, male, aged 50 years, had a hardness of hearing which began gradually about thirty years earlier There had been a continuous decrease in hearing. No tinnitus was present. The condition had been diagnosed as otosclerosis. (2-7) The others (*III 2*) in this fraternity had normal hearing.

Pedigree 2 (fig. 58, Gray,[35] 1917, table V).—The father (*II 1*) was not deaf. The earlier family history is not known.

The mother (*II 2*) was not deaf. Her mother (*I 2*) was very deaf at 70 years of age and probably for a considerable time earlier. The nature of the deafness was not ascertained.

Children (6): (1) *III 1*, male, at the age of 60, had a deafness which had gradually increased since it first appeared at 40 years. Paracusis and tinnitus were present; no suppuration was reported. (2) *III 2*, male, at the age of 57, had affected hearing in both ears. Deafness began gradually without known cause at the age of 31, and rapidly became distinctly worse. Tinnitus was marked ten or fifteen years before this report, but had diminished. Paracusis was marked for fifteen or twenty years. This patient was seen by the author, who diagnosed the condition as otosclerosis. By a woman (*III 3*) with normal hearing he had 5 children (*IV 1-5*), from 18 to 5 years of age. One girl (*IV 3*), at 14 years, was observed by the author, who diagnosed her deafness with tinnitus as otosclerosis. Two children (*IV 4* and *5*), 7 and 5½ years of age, were also seen, but had no loss of hearing. (3) *III 4*, female, at the age of 52, showed deafness, first noticed at 30 years. Tinnitus and paracusis were both present. (4) *III 5*, female, at the age of 46, showed no abnormality in hearing. (5) *III 6*, female, aged 45, showed no abnormality in hearing. (6) *III 7*, male, aged 42, showed a deafness associated with tinnitus, which began a year before and was confined to one ear. The onset was gradual, without pain or known cause.

Pedigree 3 (fig. 60, Gray,[35] 1917, table VII).—The father (*II 6*), who died at 70 years of age, had normal hearing. None of the members of his fraternity suffered from deafness. His gametic formula was probably xA, *Y*A.

The mother (*II 7*) died at the age of 74. Neither she nor her brother was deaf. Their mother (*I 4*), however, had dull hearing at 50 years and probably some time before. The nature of this deafness cannot be ascertained; there is no proof that it was otosclerosis. The formula of *II 7* was probably Xa, xa.

Children (11): These ranged in age from 50 to 33 years. Six daughters (*III 1, 2, 4, 9, 10* and *11*) and 1 son (*III 5*) had normal hearing. One son (*III 3*), now dead, began to grow deaf at the age of 26, without any known cause. He suffered from tinnitus and exhibited paracusis. The condition was diagnosed as "chronic dry catarrh," a term which included otosclerosis. The eldest daughter (*III 6*), at 42 years of age, had a deafness which began at 29 years and was associated with marked tinnitus. Her twin brother (*III 7*) also began to be deaf at 19 or 20 years. Tinnitus and paracusis were present. The remaining sister

48. Albrecht, W.: Arch. f. Ohren-, Nasen- u. Kehlkopfh. **110**:15, 1922.

(*III 8*), 41 years old, had a deafness which was first noticed at 24 years. Paracusis was present. On examination by Gray, she showed typical symptoms of otosclerosis.

Comment.—By hypothesis, half of the sons and half of the daughters should show otosclerosis. Two of 8 daughters and 2 of 3 sons were found to be otosclerotic.

PEDIGREE 4 (fig. 63, Gray,[35] 1917, table XIII).—The father (*II 1*) had normal hearing. He had no recorded siblings, and his parents had normal hearing. However, his mother had 2 otosclerotic grandchildren (*III 5* and *7*) by a second marriage.

The mother is not shown in the pedigree; she presumably had normal hearing.

Children (4): (1) *III 1,* female, had deafness diagnosed by Gray as otosclerosis. (2) *III 2,* female, had normal hearing. (3) *III 3,* male, had deafness diagnosed by Gray as otosclerosis. (4) *III 4,* female, had normal hearing.

Comment.—It is probable that the father had gametes xA, *Y*A, while the mother's gametes were probably Xa, xa. On this hypothesis half the sons and half the daughters should be affected. The expectation is realized as closely as possible.

PEDIGREE 5 (fig. 63, Gray,[35] 1917, table XIII).—The father (*II 2*) had normal hearing. He was a half brother of *II 1,* referred to in the preceding paragraph.

The mother is not shown in the pedigree; she presumably had normal hearing.

One son (*III 5*) had deafness diagnosed as otosclerosis. He was the father, by a woman with normal hearing (*III 6*), of 5 otosclerotic children (*IV 1, 5, 8, 10 and 11*).

PEDIGREE 6 (fig. 63, Gray,[35] 1917, table XIII).—The father is not given in Gray's table. He presumably had normal hearing.

The mother (*II 5*) had normal hearing. One normally hearing brother (*II 2*) had an otosclerotic child (*III 5*). There was also a half brother (*II 1*), son of this mother's mother by another husband, 2 (*III 1* and *3*) of whose 4 children were regarded as otosclerotic.

The only child, a daughter (*III 7*), had deafness diagnosed as otosclerosis.

PEDIGREE 7 (fig. 65, Gray,[35] 1917, table XV).—The father (*III 2*) had normal hearing. No defects in hearing were recorded in his 2 siblings (*III 1* and *3*) or in his immediate ancestry.

The mother (*III 5*) and her 3 siblings (*III 4* and *6*) had normal hearing. No defects in hearing were described in her immediate ancestry.

Children (10): Four daughters (*IV 1, 3, 6* and *7*) and 3 sons (*IV 2, 4* and *10*) were unaffected. Three daughters (*IV 5, 8* and *9*) had otosclerosis in typical form, as diagnosed by Gray.

Comment.—Since none of the 3 males was affected, the father's gametic formula was probably Xa, *Y*a, and the mother's formula, xA, xa. On this hypothesis, half of the daughters and none of the sons should show otosclerosis. The 3:7 ratio of the daughters approaches expectation.

PEDIGREE 8 (fig. 67, Haike,[33] 1927-1928, pedigree Sa.)—The father (*III 2*), at the age of 82, had normal hearing. One of his brothers (*II 7*) became hard of hearing at 20, and was clearly otosclerotic. One sister (*II 9*) was the progenitor of 3 daughters (*IV 29-31*) with otosclerosis.

The mother, not charted, probably had normal hearing.

Children (10): There were 6 sons and 4 daughters: One son (*IV 9*), aged 46, had progressive deafness on both sides, which started at about the age of 18.

The author's diagnosis was otosclerosis. The second son (*IV 10*), aged 44, had had a hearing defect in the left ear for a year. The author diagnosed the condition as otosclerosis.

PEDIGREE 9 (fig. 67, Haike,[33] 1927-1928, pedigree Sa).—The father, not shown in the pedigree, was presumably normal. Nothing was known of the relatives' hearing.

The mother (*III 9*) had normal hearing. One of her brothers (*III 7*) died at 75 years of age. He had a hardness of hearing which was progressive from his twentieth year and became extreme before his death. The diagnosis of otosclerosis was based on histologic examination. Their mother (*II 1*) was hard of hearing.

Children (4): (1) *IV 29*, female, was hard of hearing. (2) *IV 30*, female, was hard of hearing. (3) *IV 31*, female, had had dulness of hearing since the age of 20; it was worse after childbirth and rapidly progressive. The deafness and ear noises increased after trouble with the teeth. Haike's diagnosis was otosclerosis. (4) *IV 32*, male, had normal hearing.

PEDIGREE 10 (fig. 67, Haike,[33] 1927-1928, pedigree Sa).—The father is not shown in the pedigree. He presumably had normal hearing.

The mother (*III 19*) had normal hearing, like her brother (*III 20*) and her sister (*III 18*). Her father (*II 3*) died at the age of 74. He had 2 brothers (*II 2* and *5*) and 1 sister (*II 1*) who were hard of hearing.

Children (2): (1) *IV 44*, female, had normal hearing. (2) *IV 45*, female, had hardness of hearing, probably otosclerosis.

PEDIGREE 11 (fig. 68, Haike,[41] 1928, pedigree Ma).—Father is not represented in the pedigree. He was presumably normal.

The mother (*II 4*) had normal hearing. She had a brother (*II 3*) who became hard of hearing in his fiftieth year. The nature of his deafness could not be determined.

Children (8): These ranged in age from 50 to 30: Seven (*III 5*) had sound hearing. One daughter (*III 4*) became hard of hearing at 20 years of age, and the defect progressed considerably after pregnancy.

PEDIGREE 12 (fig. 69, Haike,[41] 1928, pedigree Ri).—The father is not shown in the pedigree. He was presumably normal.

The mother (*II 3*), at the age of 76, had normal hearing, as did her parents, who died at the ages of 50 and 86 respectively.

Children (8): Four sons (*III 2*) and 3 daughters (*III 4*) were normal. One daughter (*III 3*), at the age of 36, had dulness of hearing in the left ear of several years' standing; six months earlier she had a nervous breakdown with considerable deterioration. Diagnosis of tubular catarrh on the right side was made. The patient was again examined at the age of 56 years. For nine years she had heard poorly, and in the last year had gradually grown deafer. The diagnosis was otosclerosis.

PEDIGREE 13 (fig. 69, Haike,[41] 1928, pedigree Ri).—The father (*II 6*), a brother of the mother in pedigree 12, had normal hearing at the age of 78.

The mother (*II 7*) has normal hearing. No deafness was known in her father (*I 3*), mother (*I 4*) or her mother's 5 siblings (*5-7*).

Children (4): (1) *III 5*, male, had normal hearing. (2) *III 6*, female, at the age of 49, had a deafness which had intermittently progressed since the age of 15 years. At first it was accompanied by frequent light dizzy spells, which later ceased. She suffered much from frequent headaches. The probable diagnosis was

bilateral otosclerosis, with degeneration of the labyrinth on the left side. (3) *III 7*, female, had normal hearing. (4) *III 8*, female, had normal hearing.

PEDIGREE 14 (fig. 71, Haike,[41] 1928, pedigree Be).—The father (*II 15*) had normal hearing, like his brother (*II 2*), who, married to an otosclerotic woman (*II 3*), had 4 otosclerotic sons (*III 3-6*).

The mother (*II 16*) had normal hearing. She had 1 normal brother (*II 17*) and 2 normally hearing sisters (*II 18* and *19*). Her brother had 3 offspring by an otosclerotic wife (*III 17*), 1 of whom (*IV 7*) was otosclerotic.

Children (6): Four sons (*III 27* and *29*) had normal hearing; 1 daughter (*III 28*) heard normally. Another daughter (*III 30*), at the age of 66, was hard of hearing (otosclerosis?). She had had tinnitus since her fourteenth year. The defect in hearing increased after her second childbirth, at 32 years, and still more after the third. She had 3 children, aged from 30 to 40, who had normal hearing.

PEDIGREE 15 (fig. 88, Undritz,[45] 1928, no. 38).—The father (*II 1*) had normal hearing. His mother (*I 2*) was otosclerotic. His gametic formula was probably Xa, *Y*a.

The mother (*II 2*) had normal hearing. Her sister (*II 3*) was otosclerotic. The mother's gametic formula was probably xA, xa.

The only child, a daughter (*III 1*) was otosclerotic. Her zygotic formula was probably xX Aa.

III. *New Pedigrees: Families with Affected Children; Children Both Over and Under Thirty Years of Age.*—PEDIGREE 1 (F. H. 4).—The father (*II 4*) has normal hearing, like his 5 brothers. His gametic formula is probably Xa, *Y*a.

The mother (*II 3*), aged 64, has normal hearing, like her only sister (*II 2*). Their mother (*I 2*), now dead, is said to have been hard of hearing. The gametes of *II 3* are probably xA, xa.

Children (10): The ages of the 3 sons and 7 daughters range from 45 to 22 years. The eldest daughter (*III 7*), at 45 years, showed hardness of hearing which was worse in the left ear, where it was first noticed at about the age of 30 years. Tinnitus was violent at first. The Rinne test gave negative results. The lower tone limit, left and right, is 256. Dr. Duel, who saw this patient, diagnosed the condition otosclerosis. The patient is liable to colds. She acquired false teeth at the age of 45. The adenoids were removed at 19 years. Another daughter (*III 10*), 39 years old, has a hardness of hearing, first noticed in the right ear at the age of 24 and in the left ear at 30. It is worse in the right ear and has been ascribed to "catarrh." She rarely has headaches, but has had frequent dizzy spells since about 30 years of age. Her tonsils were removed at the age of 33. She has occasional colds and a large amount of dental repair. The Rinne test gave negative results. The lower tone limit, left and right, is 128. The patient was seen by Dr. Duel. A son (*III 13*) has impaired hearing in the left ear owing to cerebrospinal meningitis, which left him paralyzed.

Comment.—None of the 3 sons and only the oldest 2 of the 7 daughters are affected with otosclerosis. Since the onset in these daughters was at about 31 or 32 years of age, it seems probable that some of the 4 daughters under 30 years of age may show the defect in hearing later.

PEDIGREE 2 (F. H. 7).—The father (*II 3*) is said to have had normal hearing like all of his 6 siblings. Their father (*I 6*), now dead, is said to have been deaf.

The mother (*II 2*) had normal hearing, like her 7 brothers, all dead.

Children (7): Two of these died young and are not shown in the pedigree. (1) *III 1*, male, has normal hearing. (2) *III 2*, female, has normal hearing.

(3) *III 4*, male, who died at 24 years of age, was slightly deaf. (4) *III 5*, male, 68 years of age, is hard of hearing. Since the date of onset is not known, he is recorded as doubtfully otosclerotic. (5) *III 7*, female, 62 years old, has a hardness of hearing which was first noticed in the left ear at about 20 years of age and in the right ear at 30 years. The deafness has been progressive. She now hears only high-pitched voices. She has migraine, as her mother had, occasional vertigo and constant tinnitus. She suffers from chronic sore throat, has frequent colds and had all the teeth removed at the age of 37 on account of ulceration (this case is from the clinic of Dr. Shambaugh).

Comment.—It is probable that the father had gametes xA, *Y*A, and that the mother had gametes Xa, xa. On this hypothesis we should expect half of the sons and half of the daughters to be affected—a result realized as closely as possible.

Pedigree 3 (F. H. 11).—The father (*III 4*), 64 years old, has normal hearing. Two brothers (*III 2* and *5*), 65 and 62 years old, respectively, are hard of hearing; the age of onset is unknown. Their mother (*II 8*), who died at the age of 89, also was hard of hearing.

The mother (*III 3*), at 65 years of age, has normal hearing. Her mother (*II 3*), who died at 73 years of age, had a deafness which progressed suddenly. The provisional diagnosis is otosclerosis.

Children (5): These range in age from 33 to about 25 years. The 3 sons (*IV 4, 6* and *9*) have normal hearing. Of the 2 daughters, the eldest (*IV 8*), aged 29, has a deafness which is worse in the right ear. It is not accompanied by headaches or vertigo, but at first was associated with slight tinnitus, which has now disappeared. The teeth are good. An audiogram showed one-half loss of hearing at pitch 1,024. Negative results were obtained in the Rinne test. This is one of Dr. Harris' patients. The diagnosis is otosclerosis.

Comment.—It is probable, from the genetic standpoint, that the father has the gametes Xa, *Y*a, and the mother the gametes xA, xa. Accordingly, half of the daughters and none of the sons should show an otosclerotic defect.

Pedigree 4 (F. H. 18).—The father (*II 3*) has normal hearing. He has 1 sister (*II 2*), 70 years old, who is deaf; the nature of her deafness is unknown.

The mother (*II 4*) has normal hearing, as her brother (*II 5*) and her 2 parents (*I 3* and *4*), are said to have had, but these relatives are all dead.

Children (10): Three (*III 1, 2* and *6*) died young. The remaining 7 range in age from about 40 to 23. The 3 sons (*III 3, 7* and *11*) have normal hearing. Of the 4 daughters, the 2 youngest have defective hearing. A daughter (*III 9*), aged 28, has hardness of hearing in both ears, first noticed at about 21. Tinnitus is present, which is worse at night and during the menstrual period. She has had abscessed teeth. The Rinne test gave negative results. Slight hyperthyroidism is present. This daughter has been a telephone operator for five years. Another daughter (*III 12*), aged 23, has a hardness of hearing that is confined to the right ear. It was first noticed at the age of 20. She has frontal headaches but no vertigo or tinnitus. The Rinne test gave negative results. Both of these patients were seen by Dr. Harris, who made a diagnosis of otosclerosis.

Comment.—The father's gametic constitution is probably Xa, *Y*a, and the mother's xA, xa. The expectation is that half of the daughters and none of the sons will have otosclerosis. Actually 2 of the 4 daughters are otosclerotic.

Pedigree 5 (F. H. 35).—The father (*II 2*) has normal hearing. His sister (*II 3*), at the age of 64, has a deafness which began at about 20 years of age and has progressed steadily. Both their parents were said to have become deaf at about 60 years of age. No other history is known.

The mother (*II 1*) is believed have normal hearing. Her family is unknown.

Children (5): These range in age from 39 to 29 years. The 3 sons (*III 1, 4* and *5*) are unaffected. One of the 2 daughters (*III 3*), 35 years of age, has a hardness of hearing in both ears which was first noticed at about 18 years of age. A possible cause that has been assigned is temporary diabetes. This daughter has a continual buzzing in the head and occasional sore throat. Her teeth are in good condition.

Comment.—Since none of the sons and half of the daughters are affected, it is probable that the father's gametic formula is *X*a, *Y*a, and the mother's xA, xa.

PEDIGREE 6 (F. H. 40).—The father (*II 11*) is believed to have normal hearing. His relatives are unknown. His gametes are probably xa, *Y*A.

The mother (*II 10*) has normal hearing, like her 2 sisters (*II 8* and *12*) and 3 brothers (*II 13, 15* and *16*), but their father (*I 3*) was somewhat hard of hearing, like 1 of his brothers (*I 2*) and his sister (*I 7*). The gametic constitution of *II 10* is probably Xa, Xa.

Children (7): Four daughters (*III 14* and *17*), from 43 to 30 years of age, and 2 sons (*III 16*) have normal hearing. The remaining son (*III 18*), 32 years of age, has a hardness of hearing which first developed at 10 years of age without assigned cause, and which is worse in the left ear. He has headaches before the onset of colds, and also suffers from tinnitus. No infections of the sinuses or nose are reported. His teeth are resistant. His zygotic constitution is probably X*Y*, aA.

PEDIGREE 7 (F. H. 44).—The father (*II 3*) had normal hearing.

The mother (*II 4*) has normal hearing.

Children (7): (1) *III 4*, female, is said to have normal hearing. (2) *III 5*, male, hears normally. (3) *III 12*, male, is possibly slightly hard of hearing, but hears over the telephone. (4) *III 15*, female, has a hardness of hearing that was first noticed at 16 years in connection with the first menstruation; no loss of hearing occurred after the last pregnancy. She has no headaches and rarely suffers from vertigo. The tonsils were removed at the age of 20, without any effect on the hearing. She has occasional sore throat. The teeth are nonresistant. The patient once had otitis media. Paracusis and loud tinnitus are present. The Rinne test gave negative results. Bone conduction is increased. Both stapes are fixed (Lewis). There is loss of hearing for extremely high and low tones. The diagnosis made at Dr. Crowe's clinic was otosclerosis. (5) *III 18*, male, aged 37, has normal hearing. (6) *III 21*, female, has a progressive deafness first noticed at 16 years. She has no headaches, vertigo, earache or sore throat. Tinnitus is present. Many teeth are filled. The Rinne test gave negative results. The Weber test was not referred. Both stapes were fixed (Lewis). (7) *III 22*, female, 18 years old, has normal hearing.

Comment.—The father's gametes are probably Xa, *Y*a; the mother's xA, xa. The expectation is that no sons but half of the daughters will be otosclerotic; 2 of the 4 daughters are.

PEDIGREE 8 (F. H. 45).—The father (*II 4*), who died at 85 years of age, had normal hearing, as did his 3 siblings and their parents, so far as is known.

The mother (*II 5*), who died at 48 years of age, had normal hearing. Five of her siblings (*II 10, 12, 14, 16 and 20*) were otosclerotic.

Children (8): One son (*III 7*) died in infancy, and a daughter (*III 12*) died at 8 years of age. The eldest son (*III 3*), aged 65, has just recently become slightly hard of hearing. The second son (*III 6*), who died at 40 years of age, was slightly hard of hearing for several years. The third son (*III 13*), who died at 68 years, showed bilateral otosclerosis on histologic examination. Of the daughters, the eldest (*III 1*) died at 65. She had been hard of hearing since 35 years of age,

and showed paracusis, headaches and tinnitus. No infection of the sinuses or throat was reported. Another daughter (*III 8*), aged 62, has a progressive hardness of hearing which began twenty or twenty-two years ago and became worse after pregnancy. This is not accompanied by headaches or dizziness, but before the deafness set in, there was rumbling and roaring in the head. The throat and nose are not susceptible to infection. The teeth decay easily. Paracusis is present. The Rinne test gave negative results. On account of the late onset, it is somewhat uncertain if the condition in this case is otosclerosis.

Comment.—The diagnosis of otosclerosis is somewhat uncertain and the symptoms are not typical. We probably have to do with a fraternity affected by otosclerosis in both sexes, which indicates a genetic formula of xA, *Y*A for the father and Xa, xa for the mother. According to this formula, about half of the children of each sex should be affected.

Pedigree 9 (F. H. 46).—The father (*VI 17*) was not hard of hearing until during the last two or three years of his life, when, in connection with his final illness, he had slight deafness. His sister (*VI 27*) has progressive deafness, which began at 25 years of age. She is now very deaf and shows paracusis. Their parents had good hearing.

The mother (*V 12*), aged 67, a distant relative of the father, has excellent hearing. She has a brother and sister who are hard of hearing, as their mother was.

Children (5): (1) *VI 6*, female, aged 41, has a deafness which was first noticed in the right ear at from 10 to 12 years of age and in the left ear at 30; it has gradually increased. She was subject to earaches in both ears as a child. She had a loss of hearing during pregnancy and has had tinnitus. Tonsillectomy has been performed. The symptoms of defective hearing were exaggerated by a bad fall at the age of 33. Paracusis was present. This is one of Dr. Duel's cases. (2) *VI 8*, female, at 39 years, has hardness of hearing with tinnitus and a negative Rinne test; she has been especially deaf in the left ear from childhood. The condition was diagnosed as otosclerosis by Dr. Duel. (3) *VI 10*, male, had good hearing. (4) *VI 12*, female, and (5) *VI 13*, male, have normal hearing.

Comment.—The 2 sons are unaffected and 2 of the 3 daughters have otosclerosis, suggesting that the gametic formula of the father is Xa, *Y*a, and that of the mother xA, xa.

Pedigree 10 (F. H. 47).—The father (*II 6*) had normal hearing at the age of 86. His father (*I 2*) is recalled as deaf and blind when he was very old. The age of onset is unknown.

The mother (*II 5*) had normal hearing. Her sister's 2 daughters were deaf. In *III 3*, deafness followed scarlet fever. In *III 4* there is a catarrhal deafness, first noticed at about the age of 40 years.

Children (4): (1-2) *III 5* and *7*, female, now dead, had normal hearing. (3) *III 9*, female, who died at 18 years of age, had normal hearing. (4) *III 10*, female, has progressive deafness, which is worse in the left ear; it was first noticed at the age of 27, after a pregnancy, but there was temporary deafness in connection with an abscess of the ear, at the age of 20. Occasional headaches occur, and, recently, some vertigo. There have been no infections of the sinuses or nose, but frequent colds and some sore throat. Tinnitus and paracusis are present. The Rinne test gave negative results. Bone conduction is shortened on the left side.

Comment.—In this nearly typical—though late appearing—case of otosclerosis, the parents themselves were unaffected, but each had deaf near-relatives, whose deafness was not, however, typically otosclerotic. Owing to the death of all ante-

cedents, details are wanting. If the father had gametes xA, *Y*a and xa, *Y*a, and the mother gametes Xa, xa, one fourth of the daughters should be otosclerotic, just as found.

IV. *Pedigrees from the Literature: Families with Affected Children; Children Both Over and Under Thirty Years of Age.*—PEDIGREE 1 (fig. 56, Gray,[35] 1917, table II).—The father (*III 4*), who died at the age of 60, had normal hearing. One sister (*III 2*) became deaf in middle life. Their father (*II 3*) and his brother (*II 1*) became deaf fairly late in life.

The mother (*III 11*) had normal hearing. However, in 2 of her siblings (*III 7* and *9*) the hearing became dull in middle life. Their mother (*II 12*) became deaf at about the age of 50. A sister of *III 11* had a child (*IV 10*) who was deaf at the age of 30.

Children (9): (1) *IV 1,* male, examined at the age of 46, had a deafness which was first noticed at 11 years; it had become more pronounced during the last ten years. Tinnitus was distressing; it was worse in the right ear, which was the better hearing one. Paracusis was pronounced. The diagnosis was otosclerosis. (2) *IV 2,* male, at the age of 44, was somewhat deaf. Deafness was first noticed at the age of 30. The diagnosis was otosclerosis. (3) *IV 3,* male, had normal hearing. (4) *IV 4,* female, underwent an acoustic examination at the age of 38. She had dulness of hearing which began in the right ear at 16 years and in the left ear at 23. Tinnitus made its appearance later, and was moderately severe. Paracusis was noticeable. The diagnosis was otosclerosis. (5) *IV 5,* male, at the age of 37, had a dulness of hearing which was first noticed at 28 or 29 years. Tinnitus was present. The diagnosis was otosclerosis. (6) *IV 6,* female, underwent an acoustic examination at the age of 36. She had a dulness of hearing that began in the right ear at 29 and in the left ear at 32. Tinnitus of a buzzing sort was constant in the right ear and rare in the left. Paracusis appeared at the age of 35. There was no history of nasal or nasopharyngeal infection, earache or otorrhea. The diagnosis was otosclerosis. (7) *IV 7,* female, had an acoustic examination at the age of 32. She had hardness of hearing which became deafness after the birth of her 2 children. Tinnitus appeared several months after the deafness; it was constant in the right ear and occasional in the left. There was no evidence of dysfunction of the semicircular canals or the vestibule. The diagnosis was otosclerosis. (8) *IV 8,* male, in an acoustic examination was found to have normal hearing in every respect. (9) *IV 9,* female, at the age of 25, had an acoustic examination, and her hearing was found to be normal in every respect. It is, of course, possible that otosclerosis may have developed at a later age in either or both of these last 2 persons, since the onset is sometimes delayed until the age of 30.

Comment.—The father's gametes were probably xA, *Y*A; the mother's gametes, perhaps Xa, xa. On this hypothesis, up to about half of the sons and half of the daughters should be affected. Three of 5 sons and 3 of 4 daughters were already affected. The proportion is slightly greater than expectation but is readily accounted for by an abnormal chance run.

PEDIGREE 2 (fig. 68, Haike,[41] 1928, pedigree Ma).—The father (*II 2*) was thought to have normal hearing. A brother (*II 3*) became hard of hearing in his fiftieth year, but the kind of deafness cannot be determined.

The mother (*III 6*) was a niece of her husband. An uncle (*II 3*) was hard of hearing, and her first cousin (*III 4*) had a hardness of hearing at the age of 20, which progressed after pregnancy; this was probably otosclerosis.

Children (9): (1) *IV 1,* female, had normal hearing. (2) *IV 2,* male, had normal hearing. (3) *IV 3,* male, had a hardness of hearing which was first

noticed during the sixteenth year. (4) *IV 4,* male, had a hardness of hearing which was first noticed after the sixteenth year. (5) *IV 5,* female, at the age of 66, had a hardness of hearing on both sides, the onset of which was at about 20, following diphtheria. Deafness became noticeably worse after 4 childbirths. At 40, she heard conversation with difficulty; at 45 years, only with the help of an ear trumpet. The author's diagnosis (as a result of clinical examination) was otosclerosis. (6-7) *IV 6* and *7,* female were normal. (8) *IV 8,* female, died of heart disease at the age of 9 years. (9) *IV 9,* male, was normal.

Comment.—Since both sons and daughters were otosclerotic, it is probable that the gametes of the father were xA, *Y*A, and those of the mother, Xa, xa. On this assumption one would expect half of the sons to be affected; just half of them were. Also one would expect that not over half of the daughters would be affected; 1 of 4 was hard of hearing.

Pedigree 3 (fig. 70, Haike,[41] 1928, pedigree Co).—The father (*II 4*) had normal hearing, like his 9 siblings (*II 1-3*).

The mother (*II 5*) had normal hearing, like her 6 siblings (*II 6*). This mother and father are first cousins. The common stock is insufficiently known, but no members of the family were known to be deaf, despite the fact that some of them lived to between the ages of 71 and 94 years.

Children (4): One son (*III 1*) was acoustically examined by the author at 32 years of age. He had a hardness of hearing that was first noticed in that year. Deafness was present on both sides, with tinnitus. The diagnosis was otosclerosis. The 3 younger children (*III 2-4*) were unaffected.

Comment.—Although the history is fragmentary, it seems probable that the related parents each carry a factor for otosclerosis and that the father's gametes were xa, *Y*A and the mother's factors Xa, xa.

Pedigree 4 (fig. 72, Haike,[41] 1928, pedigree Th).—The father (*III 3*), who died of apoplexy of the brain, was believed to have been of normal hearing, as were 2 siblings (*III 1 and 2*).

The mother (*III 4*) was believed to have normal hearing; her 2 siblings (*III 5 and 6*) and her parents (*II 1 and 2*) were likewise believed to hear normally.

Children (9): (*1-4*) Four daughters (*IV 2 and 3*) had normal hearing. (5) Another daughter (*IV 4*), acoustically examined at the age of 37, had had progressive deafness for several years; since 19 years of age, she had had tinnitus, produced only on excitement. The diagnosis made by Haike was otosclerosis. (6) A son (*IV 5*) had hardness of hearing diagnosed as otosclerosis by another physician. (6-9) There were 3 other children (*IV 6*) presumably younger; their age and sex are unknown.

Comment.—Since children of both sexes were otosclerotic, it seems probable that the paternal gametes were xa, *Y*A and xA, *Y*a and the maternal gametes Xa, xa. On this hypothesis half, or a smaller proportion, of the sons and daughters should be affected with otosclerosis. The proportion is less than half.

Pedigree 5 (fig. 75, Hammerschlag,[49] 1906, his fig. II).—The father was not charted. He probably had normal hearing. His relatives were unknown.

The mother (*III 2*) had normal hearing. Her father (*II 2*) was hard of hearing like his brother (*II 1*) and brother's son (*III 1*).

Children (9): (1) *IV 1,* female, was believed to be otosclerotic. (2) *IV 2,* female, in an acoustic examination at the age of 35, showed a hardness of hearing

49. Hammerschlag, V.: Vererbbarkeit der Otosklerose, Monatschr. f. Ohrenh. **40**:443, 1906.

which began in girlhood. It had been continuous, symmetrical and progressive. Tinnitus was present. The results of the Rinne test were negative. (3-4) Two children, *IV 3*, female, showed normal hearing. (5) *IV 4*, male, had a hardness of hearing believed to be otosclerosis. (6-9) The remaining children, *IV 5*, were believed to have normal hearing.

Comment.—Since both sons and daughters were affected, it is probable that the paternal gametes were xA, *Y*a; xa, *Y*A; the maternal gametes, Xa, xa. It is possible that some of the younger children later showed otosclerosis. At the time of the report 1 of the 5 sons and 2 of the 4 daughters were affected.

PEDIGREE 6 (fig. 76, Hammerschlag,[32] 1908, pedigree IX).—The father is not shown in the pedigree. He presumably had normal hearing. The family is unknown.

The mother (*III 1*) had normal hearing. Two of her siblings (*III 2* and *5*), her mother (*II 1*), her mother's sister (*II 2*) and 1 first cousin (*III 7*) were hard of hearing, her 2 siblings in 1 ear and the other 3 relatives in both ears.

Children (10): (1-5) Five children of both sexes (*IV 1-3*) had normal hearing. (6) *IV 4*, male, was acoustically examined by the author at the age of 32. One ear was more affected than the other, in which the hearing had been subnormal only during the last two years but was now getting worse. The diagnosis was typical otosclerosis. The remaining 4 children were unaffected.

Comment.—Since none of the 3 daughters was affected, it is possible that the paternal gametes were xa, *Y*A and the maternal gametes Xa, xa. One half of the sons should have been affected; only 1 out of 3 of the older sons were. The 3 younger ones may have been below the age of incidence.

PEDIGREE 7 (fig. 78, Kompanejetz,[50] 1930, cases 1 and 2).—The father (*II 1*) had normal hearing, but blue sclerotics and a tendency to fractures. In this respect he was just like his mother (*I 2*).

The mother (*II 2*) had normal hearing. Her relatives were unknown.

Children (5): (1) *III 1*, male, was acoustically examined by the author at the age of 34. He had a hardness of hearing which began at the age of 22. There was periodic tinnitus. The results of the Wassermann and Rinne tests were negative. Bone conduction was prolonged. The diagnosis was otosclerosis. He had had 4 fractures and showed blue sclerotics. (2) *III 2*, male, acoustically examined by the author at the age of 28, had a hardness of hearing that had been noticed for one and a half years. The Wassermann test gave negative results; the Rinne test, positive. The diagnosis was otosclerosis or involvement of the cochlear nerve. The patient had had 4 fractures and showed blue sclerotics. The remaining 3 children were unaffected.

Comment.—The father may have had the gametic constitution xa, *Y*A and the mother Xa, xa. This would produce only defective sons. The association with osteopsathyrosis and blue sclerotics which has been noted in other pedigrees of this series is of interest.

As one examines the foregoing family histories, one is impressed by the fact that, though these parents of otosclerotic children have normal hearing, in most cases 1 or both of them have close relatives with hardness of hearing—probably otosclerosis. In the case of so rare a disease as otosclerosis, this indicates that a genetic factor is present. Such a

50. Kompanejetz, S.: Zur Frage über die Ursachen der Schwerhörigkeit bei blauen Skleren und Knochenbrüchigkeit, Monatschr. f. Ohrenh. **64**:193, 1930.

condition is expected in the case of a recessive trait. It is also found when the appearance of a trait depends on the cooperation of two factors. In such cases one of the two factors, alone insufficient to cause otosclerosis, may be carried in each parent and be combined in the progeny. The existence of affected close relatives of the unaffected parents is an indication that the factors of otosclerosis are in the parental stocks.

E. ADDENDUM

Ten pedigrees are added here, separately, since their classification is quite doubtful. They are instructive, since some of them bear on some theories of otosclerosis which make it genetically akin to hardness of hearing. One is a history in which most of the progeny are too young for full diagnosis.

PEDIGREE 1 (F. H. a).—The father hears normally. He has a sister who had tinnitus during her early twenties; progressive deafness came on with asthma at about the age of 50 and was diagnosed as catarrhal and nervous. Tinnitus is present. She has no headaches, vertigo, infection of the sinuses or nose, or sore throat, but many colds; at present she is suffering from asthma. The Rinne test gave negative results; bone conduction was somewhat shortened. The condition is probably a slowly developing otosclerosis complicated by asthma.

The mother has a progressive deafness that was first noticed at 30 and which affected especially the right side. She has continuous tinnitus, which is lessened at present; she also suffers from headaches, vertigo causing falls and occasional sore throat. The teeth decay easily. There is no goiter and the ears are dry. Paracusis is present. The Rinne test gave negative results. Her father and her father's sister were hard of hearing for years before their death.

Children (3): A daughter had progressive hardness of hearing which began at the age of 20, without assignable cause. She has no history of infections of the sinuses or nose and rarely has sore throat or colds. Her teeth decay easily. There are slight tinnitus and paracusis. The Rinne test gave negative results; hearing was limited for both high and low tones. Both stapes are fixed (Lewis). The hardness of hearing is probably otosclerotic in origin. Another daughter, aged 22, and a son, aged 12 years, have normal hearing.

Comment.—This pedigree shows a typical case of otosclerosis in the offspring of an otosclerotic mother, whose father's sister was also affected. The fraternity is too young to show potentialities.

PEDIGREE 2 (F. H. b).—These data are referred from the Johns Hopkins Hospital. The propositus died of a tumor of the brain at the age of 47. She became hard of hearing in the right ear at 35 years of age and in the left ear at 41. Her hearing was seriously impaired at 45, and before death no vibrations above 4,096 were heard in the left ear and none above 2,896 in the right. The Rinne test gave positive results, but both air and bone conduction were very short. Sections of the temporal bone made after death showed that deafness was due to otosclerosis with complete ankylosis of the stapes foot-plate (only the right bone was examined). The sister of the propositus was slightly hard of hearing for two years before her death at the age of 27. A brother, who died at 25 years of age, was not hard of hearing. Their mother was somewhat hard of hearing. Her deafness started during pregnancy.

The propositus had 2 daughters. One, who died at 14 years of age, had normal hearing. The other, at the age of 28 years, has a hardness of hearing which is worse in the right ear and which was first noticed after the birth of the first child and intensified after the birth of the second. She has occasional dizzy spells, tinnitus and no paracusis. The Weber test is referred to the right (the poorer) ear. The Rinne test gave positive results on the left; negative, on the right. Otoscopic examination showed the right ear drum intact and not retracted; it was slightly dull in appearance. There was a light cone, but the outlines were not as sharp or defined as in the left ear. The left ear drum was also intact and not retracted. A well defined light cone was seen; there was a slight pinkish color to the drum extending from the middle posterior portion around to the anterior inferior part. The condition was diagnosed as otosclerosis at the Johns Hopkins Hospital.

PEDIGREE 3 (F. H. c).—A fraternity of Polish Jews was seen, and some oral data were obtained. It is stated that the father, who died at 63 years of age, had a loss of hearing of about 15 per cent. None of his relatives was known. Of the mother it is stated that she had normal hearing like her 4 brothers and her parents. It is quite uncertain whether or not the father had otosclerosis.

Children (9): (1) A daughter, aged 50, is hard of hearing; the onset of deafness occurred at about the age of 30, after the birth of the second child. There were no symptoms in the head, nose, sinus, tonsils or throat, but the teeth were nonresistant. (2) A son, at the age of 49, had a hardness of hearing which was first noticed at 25 years; the mucous membranes of the head were resistant. (3) Another son has no ear trouble. (4) A daughter, aged 46, has a hardness of hearing which was first noticed at 30 years of age, after the birth of her first child. She had had no infections of the head. The loss of hearing is about 35 per cent. (5) A daughter began to be deaf about the age of 36, following the birth of her third child. The membranes of the head are susceptible to infection. The loss of hearing is about 4 per cent. (6) A daughter has a slight hardness of hearing that began at about 35 years of age. There are no symptoms of the nose or throat. (7) Another daughter has normal hearing. (8) A son, aged 38, has a 40 per cent loss of hearing, the onset of which was at the age of 24. Tinnitus is also present. The nose and throat are fairly resistant. The trouble started while the patient was working in a factory where the noise was "terrific." (9) A daughter, aged 35, has a loss of hearing which has been estimated at 35 per cent. The onset occurred at 11 years of age, when menstruation began. It is worse during pregnancies.

PEDIGREE 4 (F. H. d.)—This material was referred by Dr. Dench. The father is said to have been slightly hard of hearing. One of his sisters is said to be deaf, and a brother had a deaf daughter. The father's mother had a sister who was a deaf mute.

The mother's father had 3 nephews and nieces who have, or had, defective hearing.

Children (7): (1) A son had a hardness of hearing that was first noticed at 40 years of age; he has some tinnitus but few headaches. There is no history of vertigo or infection of the nose or sinuses. Several teeth were replaced at 55 years. (2-3) Two sons have normal hearing. (4) Another son is hard of hearing. (5-6) Two sons are not known to be deaf. (7) A daughter has a progressive hardness of hearing that is worse in the left ear; it was first noticed at 13 years and is accompanied by tinnitus. She has few headaches, but frequent heavy colds and occasional sore throat.

PEDIGREE 5 (F. H. e).—The father hears normally but has a sister who, at 61 years, had a hardness of hearing which was slightly noticeable while she was in school and has progressed since. She is susceptible to headaches, colds and sore throat. The diagnosis is chronic labyrinthitis. The lower tone limit is 26; the upper tone limit, 256; this improves after inflation. A brother of the father, at 74, is said not to be deaf. Another brother became deaf at 8 years of age, following scarlet fever; the deafness gradually progressed during college days until a tennis ball struck the ear, causing a hemorrhage there. It progressed rapidly after that, and the patient was totally deaf at his death at 64 years of age. A third brother became deaf in the right ear at the age of 10 years, and in the left ear at 54; the assigned cause was scarlet fever. A constant discharge occurred from the right ear. Frontal sinusitis was present. "Exostosis on the inferior wall of the right meatus is due to inflammation. An operation on the right ear showed the bone to be entirely 'crumbly.'"

The mother hears normally, as do her 2 siblings and their parents.

Children (2): (1) One daughter is hard of hearing in both ears. The onset occurred at 12 years of age; the loss was greater for the low notes. The inner wall of both ears is pink. The results of the Rinne test were negative. Bone conduction is increased. There are occasional headaches, but little tinnitus, vertigo or infection of the nasal mucosa. (2) The other daughter has normal hearing.

Comment.—The father's gametes are probably xA, *Y*A; the mother's, Xa, xa. By hypothesis, half of the daughters and half the sons should be otosclerotic; half of the daughters and half of the sons are. The testimony concerning the exostoses and defective bone formation in the third brother of the father is noteworthy.

PEDIGREE 6 (F. H. f).—Only 2 in the family are reported to have a hearing defect; this has not been fully investigated.

A woman, aged 21, first noticed deafness at 12 years of age, when she was in the fifth grade. The defect has made no marked progress in this woman, who is now married. The teeth are unresistant. Headaches and vertigo occur, but are not marked. There is complete loss of hearing for low tones in the right ear.

Her father is the only other person in the pedigree marked as having defective hearing. He is a physician, aged 52. His defect was first noticed at 44 years of age (an age when labyrinthine deafness is common). The defect progressed for six years and then improved. There is marked tinnitus. The history shows chronic tonsillitis but no otorrhea. The teeth are very good (only 3 fillings). The lower tone limit is normal. The test with the Galton whistle shows diminished hearing in both ears. Bone conduction is decreased. The diagnosis is nerve deafness.

Comment.—The daughter's hearing defect is probably otosclerosis. Her father's defect is labyrinthine.

PEDIGREE 7 (F. H. g).—The propositus, a woman, aged 55, a professor, has a deafness that began at 12 years of age. She has tinnitus, but gives no history of sinus infection or sore throat.

Her mother, who died at 62 years of age, had almost daily headaches and tinnitus. The condition had not become severe at death. The age of onset is unknown.

The mother's siblings are all dead. The eldest is thought to have been deaf.

Two of the propositus' first cousins have undergone a mastoid operation; another is totally deaf in 1 ear.

Comment.—The history lacks details. The propositus' deafness looks somewhat like otosclerosis.

PEDIGREE 8 (F. H. h).—The propositus, a man aged 46, has a hardness of hearing that was first noticed at about 25 years of age. The cause is unassigned, unless it is recurrent tonsillitis. He suffers from tinnitus. The mucous membranes of the head are resistant. Three brothers are not affected.

The father, aged 70, has a hardness of hearing that came on in later life. Tinnitus is present. The mucous membranes of the head are resistant.

The father's father and his 2 brothers had a hardness of hearing that came on in middle or later life.

Comment.—Details are lacking. We may have to do with an inheritance of progressive labyrinthine hardness of hearing, though the age of onset in the propositus is rather too early.

PEDIGREE 9 (F. H. i).—The propositus, a man aged 52, gave a history of repeated abscesses of the ear, following colds. Deafness began at about 29 years of age. The throat is resistant to infection.

The father's deafness began between 30 and 35 years of age. There is no history of false teeth (details are lacking).

Comment.—It is impossible to diagnose this deafness as otosclerosis. There are 2 others with hardness of hearing in the pedigree.

PEDIGREE 10 (Gray,[35] 1917, table X).—The father had normal hearing. Nothing is known of his relatives.

The mother had normal hearing. By another husband, who also had normal hearing, she had an otosclerotic child.

The son of the aforementioned parents, who was seen by Gray at the age of 14 years, had deafness diagnosed as otosclerosis.

(To be Concluded)

IRRIGATION OF THE EAR IN ACUTE OTITIS MEDIA

IS THERE RISK OF SPREADING THE INFECTION TO THE MASTOID CELLS?

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It has been customary to treat open surface infections with antiseptic solutions. The obstetrician has treated acute infections by intra-uterine douching, but has found that such irrigations in acute infections apparently spread the infection occasionally into the peritoneal cavity. Some otologists have suspected that irrigation of the ear in acute otitis media may tend to spread infection to the mastoid. The analogy between the mastoid and the peritoneal cavity is not very close. The latter is a collapsible space, with no positive air pressure from within, controlled by the abdominal muscles from without and the organs from within. Fluid may enter readily as the abdominal muscles relax. On the other hand, the mastoid air spaces have rigid, bony walls and the entrance is from one inlet, which is also the outlet for the air. This is the aditus ad antrum. When solutions are irrigated into the middle ear they cover the only outlet for the air, and since the air cannot escape, water cannot enter.

I undertook to determine whether irrigation solutions do enter the mastoid air spaces. I selected a few cases of mastoiditis which required operation, and irrigated the ear with a 1 per cent solution of methylene blue (methylthionine chloride, U. S. P.) one hour prior to operation, using for this purpose a pint (473 cc.) of fluid from a douche bag suspended 2 feet (60 cm.) above the level of the ear. In addition, 50 cc. of the same solution was injected into the ear while the patient was on the operating table just before making the primary incision. For this purpose I used a piston syringe, employing a good deal of force. The canal was dried out thoroughly before the external plate of the mastoid was opened, and in not one instance did I find a trace of the dye, even in the most anterior cells.

Altogether this experiment was tried out on five patients:

REPORT OF CASES

CASE 1.—A boy, 4 years old, had had a running ear for three months with a perforation in the anterior segment. The treatment with methylene blue was carried out and the mastoid operated on, on May 1, 1932. The process was large and pneumatic. The spaces were filled with pus and there were evidences of necrosis. No trace of the dye could be detected.

CASE 2.—A girl, aged 11 years, had a discharging ear for two years. The perforation was in the anterior segment of the drum membrane. The ear was treated with methylene blue and the mastoid operated on, on June 6, 1932. Pneumatization was not fully developed and there were areas of sclerotic bone with islands between, where necrosis was suspected. No trace of the dye was found.

CASE 3.—In a woman, aged 22, the drum membrane had been incised on June 6, 1932; a semilunar incision was made, beginning about the middle of the posterior border and cutting downward and forward and then upward to approximately the middle of the anterior border. On June 15, after douching with methylene blue, an irrigation such as that already described was done and mastoidectomy was carried out. The mastoid process was large and pneumatic, the cells were filled with pus and necrosis was present. No trace of the dye was discovered.

CASE 4.—In a boy, aged 6 years, an incision of the drum membrane similar to that described in case 3 was carried out on June 2, 1932. After irrigation with methylene blue the mastoid was operated on, on June 11. A large pneumatic processs with cells filled with pus was opened. No trace of the dye was found.

CASE 5.—In a boy, aged 10 years, the drum membrane was incised on June 7, 1932. After treatment with methylene blue as described, the mastoid was operated on, on June 17. The process was narrow but deep, and was more diploetic than pneumatic. There were pus and necrosis, but no trace of the dye was discovered.

In addition to these operations, I carried out a similar experiment on two cadavers. The drum membrane was completely removed and 50 cc. of a 2 per cent solution of methylene blue was injected into the external canal, by means of a piston syringe. In this case the fluid was left in the canal while the mastoid process was opened, and the dye was found in the anterior cells. I believe that the solution penetrated the mastoid as soon as the cortex was opened and permitted the escape of air.

In the second experiment on the cadaver I followed the same technic, but used a little more force in injecting the fluid and dried the canal thoroughly before opening the cortex. The dye was found staining the throat, as it had passed through the eustachian tube. The mastoid cells, however, were clear. No trace of the dye was found, even in the antrum.

CONCLUSION

Even with this limited number of cases, one would seem warranted in concluding that under ordinary circumstances irrigation of the ear is entirely safe so far as carrying infection back into the mastoid is concerned. A corollary would seem to be that it is doubtful whether extension of infection from the tympanum to the mastoid occurs as the result of the pus finding its way from the tympanum into the mastoid cells by direct extension.

422 Broadway.

SOLITARY NEUROFIBROMA OF THE PHARYNX

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Tumors originating from the nervous system or containing nerve elements and presenting within the oral cavity or pharynx primarily are rarely encountered. Neurofibromas developing about the face and neck most frequently occur in association with generalized neurofibromatosis (von Recklinghausen's disease). Solitary neurofibromas are unusual in these regions. Developing in the submaxillary region, they may secondarily bulge into the floor of the mouth or base of the tongue. Arising in the neck, from the spinal nerves or nerve roots, the cervical sympathetic system or the cervical portion of the cranial nerves, they may in an advanced stage encroach on the pharynx. Primary pharyngeal neurofibromas developing either as a manifestation of von Recklinghausen's disease or as a solitary tumor are extremely rare, the latter more so than the former. A review of the literature has failed to reveal another case of solitary neurofibroma primary in the pharynx similar to that presented here.

A clinical and pathologic study, made by New and Childrey,[1] of sixty-three cases of benign tumor of the pharynx and tonsil, observed in the Mayo Clinic from 1917 to 1930, did not include a single case of neurofibroma primary in this region. However, the records of the clinic contain several cases in which solitary neurofibroma of the submaxillary region and neck secondarily bulged into the oral or pharyngeal cavity. Colledge,[2] in 1930, reported a case of neurofibromatosis in a woman, aged 44, in which a tumor involved the right aryepiglottic fold and filled the pyriform fossa. In 1925, Forbes [3] reported a case of plexiform neuroma of the pharynx in a girl 14 years of age. The tumor was a large, irregularly smooth mass, situated just back of the right tonsil and occupying practically half of the faucial pharynx. It was producing dysphagia and nasal obstruction. Following tonsillectomy it increased rapidly in size, and a month later measured 6 cm. in

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From the Section on Laryngology, Oral and Plastic Surgery, the Mayo Clinic.

1. New, G. B., and Childrey, J. H.: Tumors of the Tonsil and Pharynx (Three Hundred and Seventy-Five Cases): I. Benign Tumors (Sixty-Three Cases), Arch. Otolaryng. **14**:596 (Nov.) 1931.

2. Colledge, L.: Two Tumors of the Peripheral Nerves, J. Laryng. & Otol. **45**:409 (June) 1930.

3. Forbes, H. H.: Plexiform Neuroma of the Pharynx, Tr. Am. Laryng. A. **47**:77, 1925.

diameter. It was removed under general anesthesia. The presence of numerous, irregularly pigmented areas on the patient's skin, present since birth and measuring as much as 4 by 6 cm., and of similar areas on her father's skin, indicated the probable relationship of this tumor to general neurofibromatosis. Meeker,[4] who reported the case from a pathologic standpoint, was unable to find any record of another plexiform neuroma involving the pharyngeal mucous membrane.

Penfield [5] has given the term "perineurial fibroblastoma" to solitary neurofibromas, and has called attention to the bewildering number of names applied to this tumor as being evidence of the confusion in the minds of the many clinicians who have studied the neoplasm. Among these names he mentioned solitary neurofibroma, neurinoma, fibroma of nerves, gliofibrosarcoma, peripheral glioma, schwannoma and tumor of the nervus acusticus.

Such tumors arise from the sheaths of nerves or from nerve roots. They are more commonly situated centrally, that is, on nerve roots, in contradistinction to the tumors of neurofibromatosis which are, as a rule, found peripherally. Occurring within the cranial cavity or the spinal canal, they arise most commonly from the root of the eighth cranial nerve, or from the roots of the spinal nerves, more commonly, the posterior roots. They occur rarely on other cranial nerves, especially the optic and trigeminal.

Grossly, the tumors are rounded and often nodular. They are encapsulated and may be either hard or cystic. Within the cranial cavity or spinal canal, they rarely attain a diameter of more than approximately 2 cm., since severe symptoms are produced early. Situated peripherally, they may grow to several times this diameter, as in the case presented in this paper. They are always attached to a nerve or nerve root, most commonly the eighth cranial nerve, the fibers of which pass on, or in, the enclosing capsule. The attachment to a nerve may not be demonstrable, owing to inadequate exposure and to the vascularity of the adjacent tissues. The color is usually distinctly brownish or yellowish, probably owing to the marked vascularity, and to the presence of blood pigment in the tissues, resulting from the degeneration commonly present.

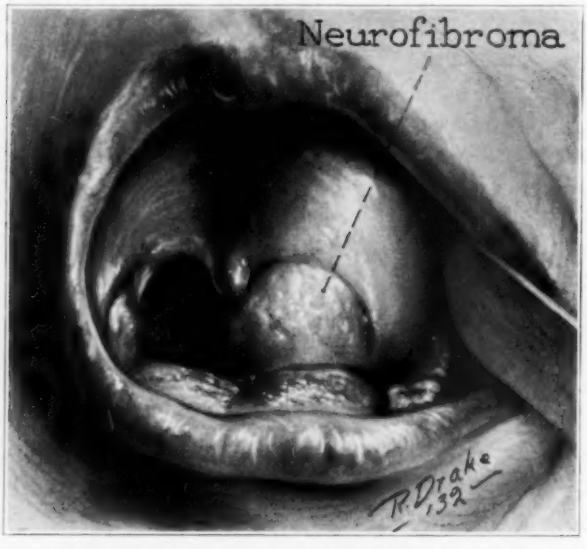
The microscopic picture of such tumors is characteristic. The outstanding histologic features are the palisade-like and parallel arrangement of the nuclei, which are usually elongated. The nuclei also tend

4. Meeker, L. H.: Plexiform Neuroma of the Pharyngeal Mucous Membrane (Ganglioneuroma), Proc. New York Path. Soc. **25**:9, 1925.

5. Penfield, Walter: The Encapsulated Tumors of the Nervous System: Meningeal Fibroblastomata, Perineurial Fibroblastomata and Neurofibromata of von Recklinghausen, Surg., Gynec. & Obst. **45**:178 (Aug.) 1927.

to be arranged in streams and eddies. Running parallel to the long axis of the nuclei are long, parallel fibers of collagen. Blood pigment is often found scattered through the tissues. Another microscopic feature that some observers have considered characteristic of such tumors is the presence of "foam cells." These are large, vacuolated, phagocytic cells present in the degenerated areas. Small fluid-filled cysts also occur in the degenerated portions of the tumor.

Perineurial fibroblastomas do not metastasize, and, in view of this, together with their histologic features, they must be classified as benign tumors. Clinically, however, even when situated peripherally they may produce severe symptoms. They are not likely to recur if cleanly



Neurofibroma of the left side of the pharynx.

removed. Such removal will, as a rule, necessitate the sacrificing of the nerve or nerve root to which they are attached.

From a diagnostic standpoint these tumors arising from the central nervous system present a problem for the neurologist. As a rule, the clinical signs and symptoms are sufficiently definite to permit of fairly accurate localization in a high percentage of cases. When they develop within the pharynx, as in the present case, or in other unusual situations, there is, of course, insufficient precedent to permit of accuracy in diagnosis. The inactivity, consistence, situation and general appearance of the tumor in the case presented in this paper were exactly those of the mixed tumors often encountered in this region, and a clinical diagnosis of probable mixed tumor had been made. At operation, the distinctly yellowish color of the tumor was striking. This was in con-

trast to the grayish white of most pharyngeal mixed tumors. The tissue was also somewhat softer and less friable than that of most mixed tumors. Without microscopic study, however, the preoperative diagnosis of mixed tumor would have been unquestioned.

REPORT OF A CASE

History.—A woman, aged 61, came to the clinic on July 13, 1931, because of frequent sore throat and persistent irritation in the throat, together with partial obstruction of the left nostril. The history was irrelevant. She had been examined by several specialists, but opinions regarding the condition of her throat had differed.

Examination.—Examination disclosed extreme prominence of the left tonsil (accompanying figure). It was firm and bulged almost to the median line of the pharynx, apparently because of marked tonsillar enlargement. In addition, a mass was palpable high in the left cervical region. General physical examination revealed well compensated, essential hypertension, but the results were negative otherwise. The blood pressure in millimeters of mercury was 190 systolic and 100 diastolic. The apparently unilateral tonsillar enlargement suggested a dyscrasia of the blood or a neoplasm. The usual laboratory examinations, including studies of the blood, gave normal results. Tonsillectomy was accordingly advised.

Operation and Course.—At operation the tonsils were found to be uniform in size, the bulging on the left being due to a large, firm, somewhat irregular mass situated external to the aponeurosis. The tumor was not incised when the tonsil was removed. Microscopic examination of the tonsils showed only chronic inflammatory change. It was now evident, also, that the mass palpable in the left upper cervical region was a direct extension of the tumor in the throat. The growth was thought to be a carcinoma of the mixed tumor type, and removal following preliminary ligation of the external carotid artery was advised. This procedure was carried out four weeks later, with the patient under intratracheal anesthesia, after healing of the tonsillar fossae was complete. The growth was found to be encapsulated, as was anticipated, but projections on its surface extended well laterally and inferiorly. The growth measured approximately 4.5 by 6 by 8 cm. Difficulty in delivering its lower lateral portion necessitated breaking into the capsule and removing a portion of the tumor with a large curet. Grossly, the tissue was of moderately firm consistence and yellowish. It appeared to be fibroelastic tissue undergoing degeneration. The capsule was then cleanly dissected out. Microscopically, the tumor was reported to be a degenerating neurofibroma.

The patient's convalescence was uneventful. In a recent communication, fourteen months following operation, the patient stated that there has been no evidence of recurrence, and that she is enjoying the best health in years.

TREATMENT OF BRONCHIAL SUPPURATION AND PULMONARY ABSCESS BY BRONCHOSCOPIC DRAINAGE

REPORT OF CASES

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The review of a series of cases of pulmonary suppuration from the bronchoscopist's experience and point of view will, I hope, add to the understanding of this disease.

The introduction of bronchoscopy initiated a new and valuable method for the diagnosis and treatment of diseases of the respiratory tracts. While a correct diagnosis is frequently made by inference, there are a number of doubtful cases that can be readily cleared up by direct observation of the tracheobronchial tree.

HISTORY

Alfred Kirstein [1] of Berlin, in 1895, introduced direct autoscopy of the larynx and trachea. Three years later, in 1898, Gustav Killian invented metal tubes and introduced bronchoscopy. Its utility was immediately recognized and numerous bronchoscopic records attest to its increasing importance. While a search of the literature does not reveal who was first to treat bronchoscopically and report abscess of the lung, I was informed [2] that Chevalier Jackson of Philadelphia inaugurated this treatment.

ETIOLOGY

The causes of abscess of the lung are not many. A foreign body may bring about pulmonary suppuration, in which the etiology is not suspected, owing to the absence of a history, and the cause is discovered only in the course of bronchoscopic inspection. The abscess which may follow pneumonia is well known, as is the tuberculous abscess. Pulmonary embolism may lead to suppuration. This has been proved experimentally (Fetterholf and Fox). Posttonsillectomy abscess due to

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From the combined services of the Beth Israel, Beth David and Sydenham Hospitals.

1. Kirstein, A., quoted by Garrison, F. H.: An Introduction to the History of Medicine, ed. 3, Philadelphia, W. B. Saunders Company, 1921.

2. Harris, M. L.: Personal communication to the author.

aspiration, while infrequent, does occur, and H. H. Vail [3] and M. C. Myerson [4] have shown the presence of blood and other secretions in the tracheobronchial tree during tonsillectomy. Even in local tonsillectomy, owing to anesthesia of the pharynx, secretions may enter the trachea during operation and become the cause of pulmonary suppuration.[5]

The inciting micro-organisms may be any of a number of bacteria which ordinarily inhabit the upper respiratory tracts. Usually the secretion from an abscess cavity contains mixed organisms and the primary invader cannot be isolated. Certain organisms (Vincent-Plaut's bacillus) may be suspected from the exceptionally offensive odor.

TREATMENT

"Early skilled bronchoscopic drainage," according to McCrae,[6] "is an invaluable method of treatment." This procedure is applicable to all pulmonary suppuration that has an outlet to the tracheobronchial tree (except tuberculosis). The earlier bronchoscopic drainage is instituted the more favorable are the results, and if an abscess of the lung is determined or suspected, the bronchoscopist's assistance should be promptly enlisted. Unfortunately, this is not being done in the great majority of cases. Some reputable internists are loath to advise bronchoscopic drainage in pulmonary abscess, whether the abscess is favorable for bronchoscopic drainage or not. Some are prejudiced because they have seen unskilled and tragic bronchoscopies performed by beginners or by mediocre endoscopists. There are others who do not favor bronchoscopies merely because they are not interested in the subject. It is true that a poorly executed bronchscopy leaves a sad impression on the mind of the bystander, but the intelligent observer should judge it with the same fairness as he would an unskilled surgical operation and not place the entire subject of bronchoscopy on the index expurgatorius.

Those who have performed bronchoscopic drainage for abscess of the lung and those physicians who have aided in or witnessed the procedure and seen the results obtained will attest to my statement that a patient with an abscess of the lung, no matter where it is located, as long as it communicates with a bronchus, should get the benefit of at

3. Vail, H. H.: The Tracheobronchial Tree in Children, with Reference to Blood Aspirated During Tonsillectomy and Adenoidectomy Under Ether Anesthesia, Arch. Otolaryng. **8**:26 (July) 1928.

4. Myerson, M. C.: Lung Abscess Following Tonsillectomy, Arch. Otolaryng. **1**:137 (Feb.) 1925.

5. Ochsner, A., and Nesbit, W.: Pulmonary Abscess Following Tonsillectomy: Preliminary Report, Arch. Otolaryng. **6**:330 (Oct.) 1927.

6. McCrae, T.: Abscess of the Lung: Medical Aspect, Arch. Otolaryng. **7**:103 (Feb.) 1928.

least six or seven bronchoscopies before he or she is surrendered to the surgeon. Should that procedure be carried out in every case early enough, surgery with its complications and great mortality would seldom be necessary. Surgery should be resorted to only in those cases in which a number of bronchoscopies were tried without improvement. Clerf reports a cure by bronchoscopic treatment of 54 per cent of the patients in a series of 121 cases. Sixteen patients showed improvement, twenty-eight were eventually referred to the surgeon and three died. Nor is Clerf the only one who reports such favorable results. The Jacksons of Philadelphia, Myerson, Kernan and Herriman of New York, Gutteridge of Australia, Martin of Edinburgh and Lemariey and Soulas of Paris have reported enthusiastically, and favor bronchoscopic drainage in abscess of the lung.

According to Jackson and his co-workers, "the theory of bronchoscopic treatment of lung suppuration is founded on impairment or inefficiency of the natural pulmonary defensive mechanism, namely, the cough reflex, the ciliary activity and germicidal action." Bronchoscopic drainage stimulates the cough reflex and restores ciliary action by removing the inspissated purulent secretion from the surface of the mucous membranes. The assertion made by some bronchoscopists, and "swallowed" by the gullible, that they insert the bronchoscope into the abscess cavity, wash out its contents and apply medication to its walls is sheer nonsense. Only when an entire lobe is involved in the abscess formation, taking in part of the main bronchus or the orifices to the secondary bronchi, is it possible to come in contact with the abscess cavity. However, such an extensive involvement speaks for pulmonary gangrene rather than for abscess. The differential diagnosis between pulmonary abscess and pulmonary gangrene and the difference in their treatment is thoroughly discussed in an excellent paper by Kline [7] of Cleveland.

In the series of cases that I am about to report, this disease either followed pneumonia or was the result of tonsillectomy in adults under general anesthesia. In my limited experience I have not treated abscess of the lung that followed tonsillectomy under local anesthesia, nor have I observed such cases in the service of others. I have, however, seen such cases reported in the literature.

REPORT OF CASES

CASE 1.—Max R., a high school boy, 17 years of age, was admitted to the Beth David Hospital on April 23, 1925, with a diagnosis of abscess of the lung following pneumonia. The subjective symptoms, physical signs, roentgenograms and characteristic sputum left no doubt as to the correct diagnosis.

7. Kline, B. S.: Pulmonary Abscess and Pulmonary Gangrene, J. A. M. A. **90**:2008 (June 23) 1928.

The boy was sinking rapidly, and his physicians, giving up the case as hopeless, called on the surgeons for advice. They, too, considered the condition too far advanced, and advised against operation. The patient was then surrendered to the bronchoscopist, and bronchoscopy was performed the same day; more than a pint of pus was evacuated from the right inferior lobe. He improved immediately. I

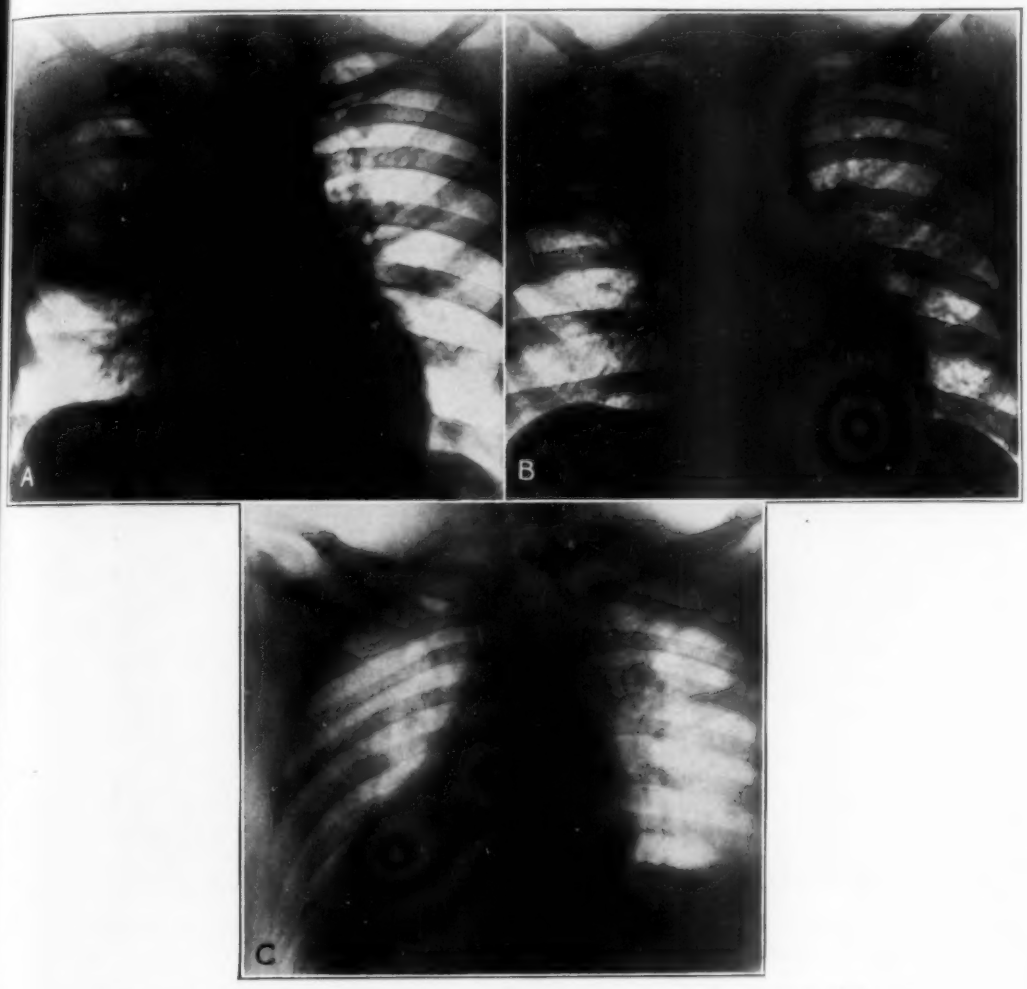

Fig. 1 (case 5).—*A*, a large encapsulated abscess of the lower part of upper lobe of the right lung. *B*, gradual improvement in abscessed area. *C*, entire absence of the lesion of the upper lobe after final bronchoscopy.

carried out six other bronchoscopies at various intervals. The patient made an uneventful recovery and was discharged as cured on August 2.

This case was described in detail in the ARCHIVES OF OTOLARYGOLOGY in 1926.

CASE 2.—In Isidore Yigdall, a painter, 40 years of age, signs and symptoms developed following pneumonia that led to a diagnosis of a right pulmonary abscess.

A roentgenogram on May 11, 1926, revealed a resolving pneumonia of the right middle lobe. By May 27 there was definite roentgen evidence of an abscess of that lobe. The patient's personal physician, Dr. Herman Pomeranz, consulted Drs. Lillienthal, Blake and James Alexander Miller, who all advised bronchoscopy.

I carried this out under local anesthesia on May 29, using a Bruening bronchoscope. The patient improved at once, expectorating with greater ease. The bronchoscopies with suction were carried out altogether six times at regular weekly intervals. Roentgenograms of the chest, taken and reported on by Dr. Maurice

Fig. 2 (case 6).—Condition reported as consolidation of the right lung, simulating collapse, due to a plug in the bronchus.

Pomeranz at regular intervals during the course of treatments, showed a gradual improvement and clearing of the right middle lobe. The patient left the Mount Morris Hospital in the best of health, and gained 49 pounds (22.2 Kg.) in a few weeks. Five weeks later he went on a three weeks' sojourn to the mountains. In the early morning before his return to the city—eight weeks after the last bronchoscopy—he was found dead in bed, death having come on him quietly without disturbance in his sleep. The cause of his death is wholly speculative. The suddenness of his death combined with the healing of the pulmonary abscess would lead one to consider an embolus from the involved area to the vital centers in the brain.

CASE 3.—Clara R., a young emaciated woman, presented herself at the bronchoscopic clinic in January, 1927. In November, 1926, a tonsillectomy had been performed under general anesthesia. Three days following operation the patient began to cough, at first nonproductively but soon with profuse foul expectoration. A diagnosis of abscess of the lung was made by Dr. Julius Miltz, who advised the patient to enter a hospital. Accordingly, she was admitted to the Mount Sinai Hospital and a diagnosis of right pulmonary abscess was confirmed. Three bron-

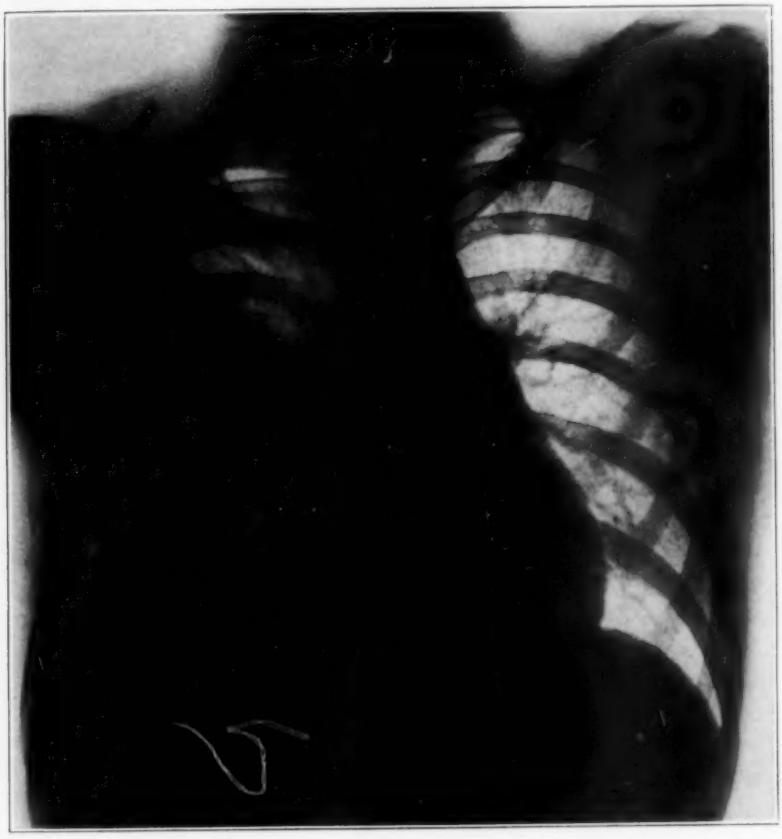

Fig. 3 (case 6).—Condition reported as perhaps an unresolved consolidation. No definite abscess formation is seen.

choscopies were performed there at weekly intervals. She then decided to leave the hospital and seek advice elsewhere.

At the clinic one could easily detect the foul odor of her breath the moment she opened her mouth. She was very pale and emaciated, coughed incessantly and brought up great quantities of foul, dark-gray sputum.

Physical signs in the right side of the chest were all characteristic of an abscess of the lung. A roentgenogram was taken and revealed a definite walled-off abscess with a fluid level in the right middle lobe.

Bronchoscopy was performed on Jan. 14, 1927, by means of the Kahler-Leiter 10 mm. bronchoscope. A large quantity of foul pus was suctioned off, and the main bronchus and orifices to the secondary bronchi were examined. The bronchial mucous membrane was intensely congested, and pus was seen to flow over the crest of the middle lobe bronchus. Seven more bronchoscopies were performed at weekly intervals, the same procedure being used. Clinical and roentgenologic indications gradually disappeared. Since then the patient has improved rapidly; she has regained her strength and remained entirely well for the past five years.

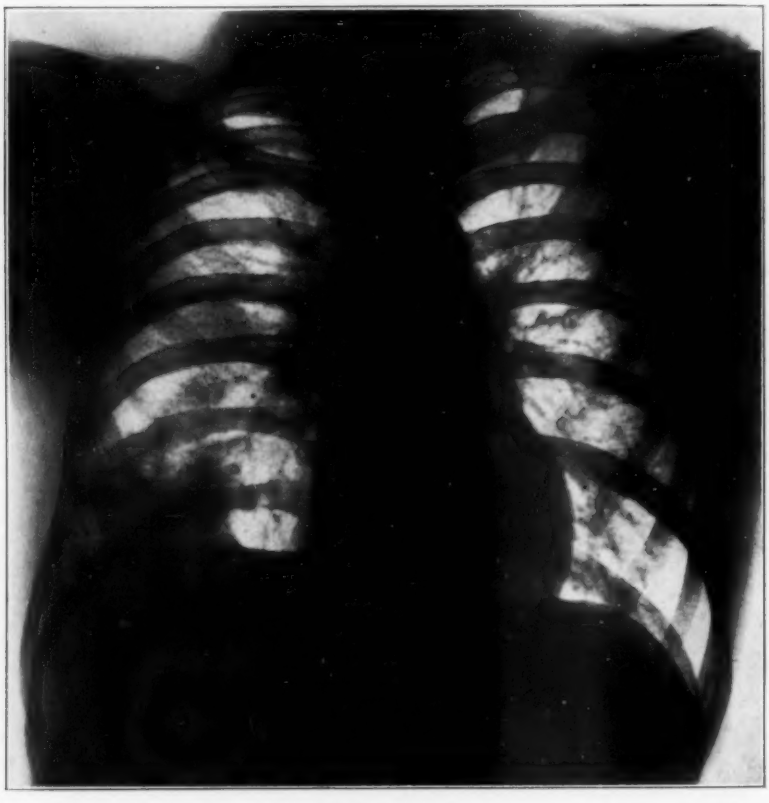

Fig. 4 (case 6).—Appearance following bronchoscopy. This was reported as marked evidence of resolution in the right lower lobe.

Case 4.—J. F., 61 years of age, was admitted to the Beth David Hospital on Sept. 7, 1929, with a history of pain in the left side of the chest, cough and expectoration of tenacious, mucopurulent, fetid and at times blood-streaked sputum of one month's duration. One year previously he had a pulmonary condition which was cured in three days.

On admission he had temperature of 101 F., and was coughing paroxysmally and expectorating profusely. Many râles were heard over the right and left lower lobes. There was slight clubbing of the fingers. Fluoroscopy showed an area of increased density in the left lower lobe. A diagnosis of pulmonary abscess was

made. The hemoglobin content was 70 per cent. Examination of the sputum was essentially negative for tubercle bacilli.

The patient was given arsphenamine until evidences of poisoning appeared as jaundice and dermatitis, which eventually cleared up. Signs in the left side of the chest cleared up, and roentgen examination showed a clearing of the lesion with fibrosis replacement. The patient stayed in the hospital for thirteen weeks. He was then discharged, but was readmitted on June 14, 1930, with a history of progressive cough and foul, profuse sputum (half a pint a day). Physical signs pointed to a lesion (abscess) in the left lower lobe. Urinalysis and examinations of the blood and sputum yielded negative results. Bronchoscopy was performed at fourteen day intervals (from June 23 to September 23, a total of six bronchoscopies). The first bronchoscopy showed the mucous membrane of the trachea to be congested and streaked with pus; the carina was thickened and congested. The caliber of the left main bronchus was narrowed and the mucosa congested, swollen and filled with foul, dark-gray, purulent secretion. The crest of the bifurcation of the inferior lobe bronchus was swollen, rigid and infiltrated. The impression that one received at the time was that of malignancy with secondary suppuration. Further bronchoscopy showed the abscess to be situated at the outer portion of the left lower lobe. Following the bronchoscopy with drainage by suction the patient felt subjectively better; cough and expectoration were diminished, the pathologic physical signs in the left lower lobe cleared up and the temperature remained even.

In this case of abscess of the left lower lobe of the lung, of unknown etiology, the patient was markedly improved by bronchoscopy and suction drainage.

Case 5.—J. M., 28 years of age, en route from Italy, was suddenly seized with a sharp lancinating pain in the lower right side of the chest, accompanied by a cough with scant expectoration. About ten days later, fever developed, and a harassing cough with a thick, tenacious, foul sputum. Physical examination, three weeks after the onset of the illness, revealed an area of marked dulness in the upper anterior and left axillary regions of the right side of the chest, with auscultation pointing to a consolidation. Examinations of the sputum failed to show tubercle bacilli, and a clinical diagnosis of abscess of the upper lobe of the right lung was made. This was corroborated by the roentgenogram, which showed a distinct encapsulated area involving the lower part of the upper lobe of the right lung, about the size of a large grapefruit, with signs of necrosis in the center.

The patient's personal physician, Dr. David H. Smith, called Dr. A. S. Elterman in consultation, and they decided to have the diagnosis verified and treatment by bronchoscopy with suction instituted.

The patient was admitted to the hospital on April 18, 1930, and I performed bronchoscopy and aspiration at weekly intervals for a total of six treatments. With these, there was a marked recession in the clinical course of the abscess, with a concomitant clearing on roentgen examination. The patient left the hospital and made an uneventful recovery.

Case 6.—Horace Dicks, a 16 year old white boy, was admitted to the Sydenham Hospital, acutely ill, on Jan. 23, 1931, with a temperature of 103 F., a pulse rate of 96, a respiratory rate of 36, physical signs of pneumonia of the entire right side and some questionable signs of fluid at the base of the right lung.

A roentgenogram taken the next day was reported as showing pneumonia of the right side. The patient's temperature was between 104 and 105 F. for seven days, and then between 102 and 103 F. for two days; then it started to assume a septic character, varying between 100 F. in the morning and 104 F. in the afternoon. This septic temperature continued for nine days. During this period some tympany

appeared over the third and fourth ribs just to the right of the sternum, and signs at the base of the right lung posteriorly began to assume characteristics of fluid. The roentgen reports on February 4 and 9 were: "Consolidation of right lung. Simulating collapse due to plug in bronchus. Fluid should be considered and ruled out in addition to the consolidation."

The temperature curve gradually declined so that it varied from normal in the morning to 100 F. in the afternoon, remaining this way for a week, during which time the physical signs seemed to clear up considerably but not completely. A

Fig. 5 (case 6).—Appearance following last bronchoscopy. This was reported as showing "recession of the abscess." The same peribronchial changes are present; they are most marked in the lower lobe.

roentgenogram at this time was reported as showing: "Marked clearing of the haziness of the entire right pulmonic field."

On February 23 the temperature again rose to 103 F., and on the next day to 104 F.; it then declined somewhat and has since varied regularly from 99 F. in the morning to 102 F. in the afternoon—an up and down type of curve.

On March 10, a needle was inserted in the chest below the scapula. No fluid was obtained. It was again inserted laterally in the lower axillary region and no fluid was obtained. On March 17 the roentgen report was: "Consolidation of the right lung. Fluid in the right pleural cavity."

On April 7, "there is marked recession of the consolidation on the right side. The entire right upper lobe is clear. There is still some involvement of the middle and lower lobes." On May 19, "the condition of the right lung is clearing. It is almost clear."

On June 10, after an injection of iodized oil, the report was: "There is no definite abscess formation. . . . One would get the impression that this is an unresolved consolidation, simulating a bronchiectatic formation."

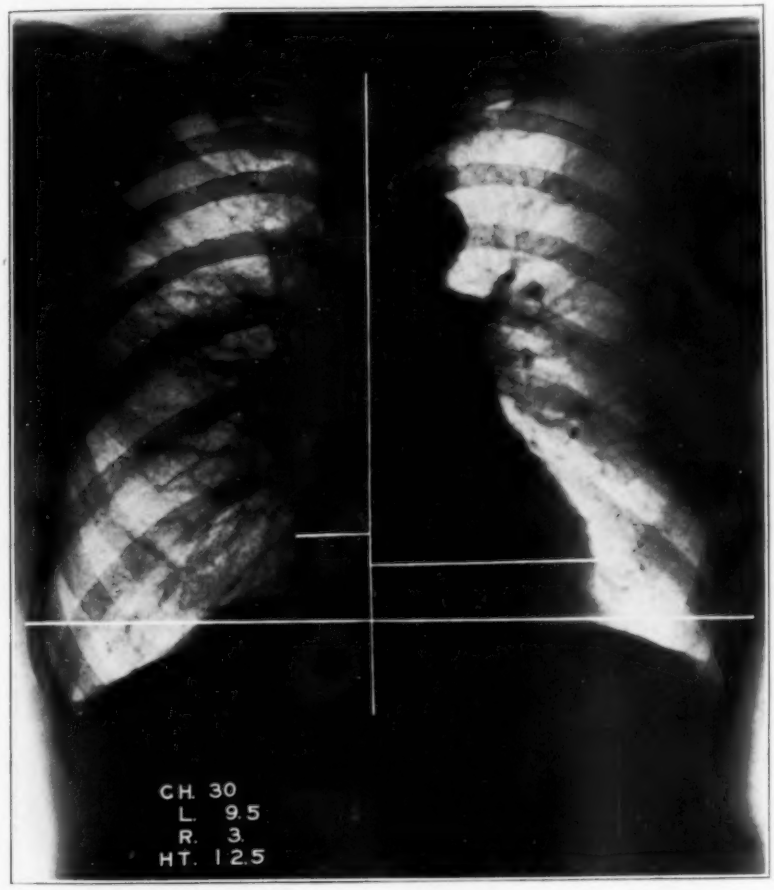

Fig. 6 (case 7).—Abscess of the middle lobe of the right lung with a fluid level. Pus is seen coming mainly from the middle lobe bronchus.

On July 1 the patient underwent bronchoscopy. "The carina was found red and edematous. The right main bronchus was covered with a thick purulent secretion which was removed by suction; the lining membrane was red and edematous. Pus was present in the entire bronchial tree below the level of the right upper lobe. This pus was aspirated."

On July 8 bronchoscopy was again performed. The condition showed marked improvement. A lesser amount of pus was present and aspirated than the week previously. A roentgenogram showed: "Marked evidence of resolution in the right lower lobe."

On July 21 the patient again underwent bronchoscopy. There was a marked improvement in the appearance of the trachea, carina and right bronchus. A small amount of pus was aspirated from the right bronchus. The roentgen report was: "Recession of the abscess. Peribronchial changes present—most marked in the lower lobe." A roentgenogram on August 20 showed no change from the appearance on July 21. Clinically the patient showed marked improvement and left the hospital quite well.

CASE 7.—Samuel Kerper was presented for bronchoscopy on July 8, 1931, at the Sydenham Hospital with a diagnosis of abscess of the right middle lobe of the

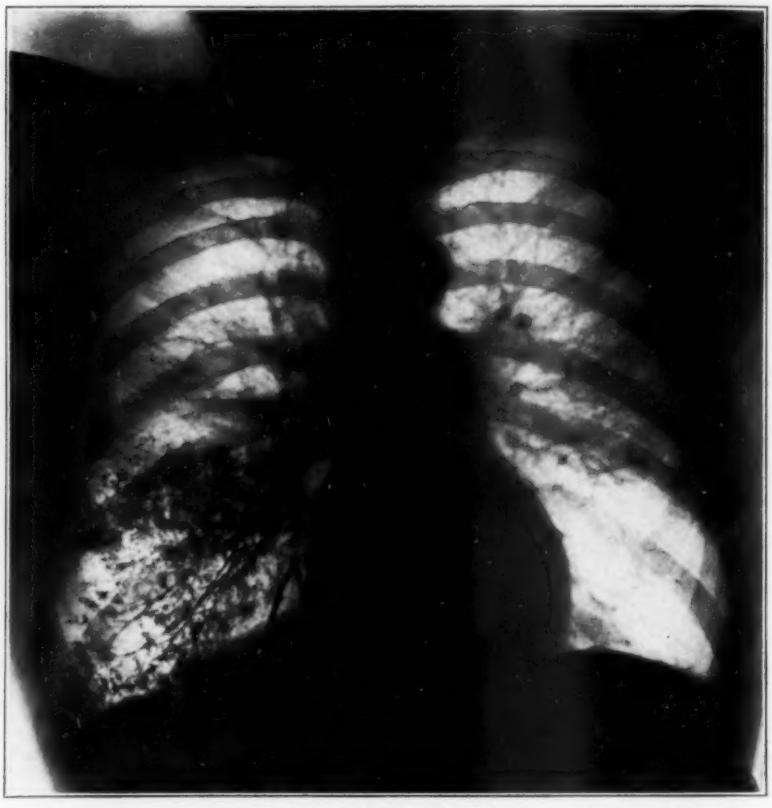

Fig. 7 (case 7).—Appearance resembles that of figure 6. The fluid level is less but still present. The inferior lobe shows the presence of the injected iodized oil.

lung. Bronchoscopy confirmed the diagnosis, and from about 4 to 5 ounces (113 to 141 cc.) of foul smelling pus was aspirated from the right lung, mostly from the region of the orifice of the right middle lobe bronchus.

The patient felt better during the next two weeks, but reappeared on July 22. A second bronchoscopy revealed a marked improvement in the appearance of the larynx, trachea and carina. In the right main bronchus there was much less pus. Suction removed from 2 to 3 ounces (70 to 85 cc.) of purulent material. The roentgen report after this treatment was: "Marked recession in the fluid present in the abscess cavity in the right middle lobe of lung."

On August 5, bronchoscopy was again performed. A general improvement in the findings of July 22 was noted. A roentgenogram taken immediately after the bronchoscopy showed "no evidence of any fluid level in the abscessed area." Clinically the patient was improved. He coughed less and the sputum was less in quantity and not foul smelling.

On August 19 another bronchoscopy was performed. The quantity of pus found was much less than on any previous examination and now centered about the opening of the right middle lobe. Roentgen examination immediately afterward revealed the same condition as on August 5, namely, an "absence of fluid level."

Fig. 8 (case 7).—The abscess cavity is clear. No fluid level is seen. The mottled area shows the remains of the iodized oil in the inferior lobe bronchi.

On August 24 the patient was seen in the nose and throat clinic, where a diagnosis of chronic sinusitis was made and confirmed roentgenologically. He received treatment there and the nasal discharge cleared up.

On September 9 bronchoscopy was again performed. Pus was found in the right main bronchus, but no pus was found to come from the secondary bronchi. The patient has made a slow but steady recovery and is now up and about.

Case 8.—L. G., 40 years of age, was admitted to the Gouverneur Hospital on Jan. 6, 1932, with the following complaint: productive cough, dyspnea and pain in the right side of the chest.

Two weeks before his admission the patient complained of pain in the chest, cough, chills, fever, weakness and dizziness. He was not confined to bed until one week before admission to the hospital, at which time he complained of a productive cough, pain in the right side of the chest, chills and fever.

Physical Examination.—The patient was apparently acutely ill, perspired profusely and had a foul odor from the mouth. Examination of the respiratory tract gave evidence of a coated tongue, carious teeth, a red throat and a foul odor from the mouth. Examination of the lungs revealed a dull area in the right axilla, extending posteriorly to the vertebral border of the right scapula and down to the angle of the scapula. Breath and voice sounds were diminished in this area, and a few moist and musical râles were present. The base of the left lung also gave evidence of dulness. Examination of other tracts yielded essentially negative results.

Laboratory Findings.—Roentgen examination revealed an abscess of the right upper lobe. On admission the urine was normal. Examination of the blood showed: white cells, 17,900; red cells, 4,000,000; polymorphonuclears, 82 per cent; hemoglobin, 80 per cent; transitionals, 5 per cent; lymphocytes, 13 per cent, and Wassermann reaction, negative. Examination of the sputum was negative for tubercle bacilli, spirilla, etc. During the first week following admission the temperature varied between 102 and 103 F.

Bronchoscopy was first performed on January 11. Under local anesthesia, with 10 per cent cocaine to which a few drops of epinephrine were added, a Kahler-Leiter 12 mm. tube was inserted through the glottis into the trachea. The lumen was adequate, the mucosa normal and the underlying cartilaginous rings visible. The carina was found in the median line and was sharp and normal. Passing to the right of the carina, a pale atrophic mucous membrane was found with the underlying cartilaginous rings standing out in great relief all along the right main bronchus. A slight amount of foul smelling secretion came from all the orifices of the bronchi of the right upper and middle lobe. The diagnosis was abscess of the lung. Weekly bronchoscopies were advised.

Between the first and second bronchoscopies, the amount of expectoration diminished from 2 cupfuls to 1 cupful of foul smelling sputum, the temperature fell from 102 to 101 F., and the patient became more cheerful.

Using the procedure described, eleven bronchoscopies were performed between January 11 and April 26. The odor diminished and the general condition of the patient improved.

On May 12, two days prior to the patient's discharge, a roentgenogram showed a clear abscess cavity. The temperature remained normal and the general condition markedly improved. The patient was advised to return to the outpatient department for observation.

Case 9.—Simon Mehlman, 52 years of age, was admitted to the hospital on March 5, 1932, with a diagnosis of abscess of the lung. On admission he complained of loss of weight for one year and cough and fever for three weeks. Three weeks prior to his admission a chill suddenly developed. Cough was very severe and the temperature 103 F.; the cough and fever continued. Foul sputum was expectorated. The patient had drenching sweats and had lost from about 10 to 15 pounds (4.5 to 6.8 Kg.) in a few months.

Physical Examination.—Positive indications in the lungs were: impairment of resonance at the left apex, anteriorly and posteriorly, breath sounds distant and numerous coarse râles in this area. There was no clubbing of the fingers, but it was difficult to evaluate the cause of the arthritic changes. The patient was expectorating foul sputum.

A roentgenogram of the chest showed an abscess cavity in the left apex. The urine was normal. The Wassermann reaction was negative. Chemical analysis of the blood gave negative results. Examination of the blood showed: red cells, 4,500,000; hemoglobin, 60 units; white cells, 16,000; polymorphonuclears, 83; lymphocytes, 17, and staff cells, 12.

Repeated examinations of the sputum gave negative results for acid-fast bacilli. The sedimentation rate was 70 per cent. Elastic tissue was found in the sputum. No spirilla were observed.

Bronchoscopy was first performed on March 15. Under local anesthesia, with 10 per cent cocaine and epinephrine, a Jackson 9 mm. tube was inserted through the glottis, into the trachea and down to the carina. The tracheal lumen was found adequate and the mucosa normal. The right main bronchus was adequate and the mucosa somewhat congested. The carina was somewhat thickened, slightly congested and resilient. The left main bronchus was filled with a foul, purulent secretion, and when this was suctioned off, pus was seen welling from the bronchus of the left upper lobe. The mucous membrane of its orifice was intensely congested and swollen. A constant escape of pus was noted. The diagnosis was abscess of the left upper lobe of the lung.

The patient continued to have a low grade fever, the temperature ranging from 99 to 103 F. The patient underwent bronchoscopy four times, the same procedure being used. Two weeks before his discharge the temperature was flat and the sputum diminished. Roentgen examination showed tremendous improvement in the left apex, with fibrosis. The patient was discharged on April 22, decidedly improved.

Case 10.—Alvina W., 34 years of age, was referred to Dr. Garlan and admitted to the Sydenham Hospital on Jan. 3, 1932.

Three weeks following tonsillectomy under general anesthesia, the patient began to have cough and expectoration. Roentgen examination of the chest revealed a circumscribed abscess in the right upper lobe with a fluid level.

Bronchoscopy was performed on January 3. Under local anesthesia, with 10 per cent cocaine to which a few drops of epinephrine were added, a Jackson 9 mm. tube was inserted through the glottis into the trachea and down to the carina. The trachea was found to be adequate and its mucosa normal. The carina was normal and resilient. On entering the right main bronchus, I found the crest of the right upper lobe bronchus very swollen, edematous and congested, and from its orifice poured forth a constant stream of offensive purulent secretion. This was drained with a suction tube, curved on its end to pass around the corner to the right upper lobe.

Three more bronchoscopies were performed at weekly intervals by the same procedure. After each bronchoscopic drainage there was marked improvement, both clinically and roentgenologically. Following the last bronchoscopy the patient made a complete recovery.

COMMENT

Since 1925 there have come to my attention twenty-six cases of pulmonary abscess. Of these patients, sixteen made a complete recovery following bronchoscopic aspiration. The number of bronchoscopies varied from three to twelve, depending on the kind, location and extent of the lesion and whether the patients came early or late for bronchoscopic drainage.

Three patients improved and were subsequently admitted to the Montefiore Hospital. Three were surrendered to the surgeon after a number of bronchoscopies were tried without the slightest result. These cases, however, were of long standing, and the patients were very septic and showed low resistance. Two of the patients died postoperatively and one recovered.

Of the remaining four patients, one disappeared during the course of treatment. Another had two bronchoscopies and improved, but the attending physician interfered by trying pneumothorax and I thought it wiser to discontinue the bronchoscopies. The other two patients died, one of them eight weeks after the final bronchoscopy. The latter case I reported, and this death should not in reality be considered as caused by pulmonary abscess.

Abscess of the lung became an important problem to the otolaryngologist in 1912, when Charles Richardson first directed attention to it as a postoperative complication following tonsillectomy. It is in these cases that early bronchoscopic drainage gives the best results. My observations are supported by similar gratifying results obtained by the Jacksons of Philadelphia, Imperatori, Kernan, Myerson, Orton and others. "There are, of course, certain fulminating cases, probably embolic in origin, in which the patient is so seriously ill that he is doomed from the onset. It is useless to attempt bronchoscopy in such patients."

Although Moore and Lukens do not advise bronchoscopy when the abscess is quite extensive or the patient moribund, I found that in at least two of my cases these contraindications did not hold true. One patient was actually in extremis (case 1) with an involvement of the entire right inferior lobe. The other (case 5) had an acute abscess, the size of a grapefruit, and he was so ill and frail that he could not help himself from the carriage to the operating table.

The actual contraindications are serious organic cardiac disease, laryngeal tuberculosis and a recent profuse hemorrhage.

Clinical Notes

AN UNUSUAL FOREIGN BODY CASE

HARRY BUTLER, M.D., BANGOR, ME.

Some months ago, I was called to see a boy (E. N.), 6 years of age, in the absence of the otolaryngologist who usually attended the family. The following history was given by the father:

Several hours before my arrival, the boy had held a pistachio nut between his teeth to crack its shell. The nut disappeared, and the boy at once became cyanotic. The father held the boy up by his feet and put his finger down the patient's throat in an attempt to dislodge the nut, or piece of nut, which he assumed to be present. The cyanosis disappeared, but as the dyspnea continued, the parents took the child to a hospital, where a house physician repeated the finger palpation, reporting later that he definitely felt something hard slip ahead of his finger. The family then returned home.

When I saw the boy, dyspnea and considerable coughing were present. It did not seem likely that more than a portion of pistachio nut shell could have passed the glottic chink, so the problem was resolved to one of two possibilties: (1) there was a portion of shell in a bronchus, or (2) an uncracked nut was lodged in the upper part of the esophagus.

The patient was admitted immediately to the hospital for roentgenologic observation and peroral endoscopic study. In the meantime, a hurried review of the available tables (Jackson) showed no report of the handling of a pistachio nut. After twenty minutes' trial with a practice board, it was revealed that the only possible instrument which would give a firm grasp, in repeated maneuvers, of such a body, which was very hard and convex both ways, was a large size, serrated-jawed, forward grasping forceps. All others slipped off, as firm pressure or traction was used.

Roentgenograms showed no evidence of a foreign body. Esophagoscopic study showed that the esophagus was free from contents as far as the cardia. Inspection of the larynx revealed that the arytenoids and epiglottis were very red and edematous. The patient's dyspnea lessened rapidly, and bronchoscopy did not seem advisable or allowable at this time, owing to the precarious condition of the larynx. The possibility of laryngeal irritation from salt, as well as from the prolonged trauma of digital exploration, was considered. The patient was returned to his room and an ice collar ordered. Within the next four days two roentgenograms were taken of the chest, and the chest was examined by an internist, who reported that there was only a slight increase in the breath sounds on the right, owing to bronchial irritation, and that it was not conceivable that these sounds were due to the presence of a foreign body.

As no proof of the presence of a foreign body in the bronchi could be obtained, the patient was discharged from the hospital, after ten days, to the care of his family physician, with a warning to the parents that on the development of cough, cough with expectoration of pus, or fever, he should be returned to the hospital at once.

I did not hear from the patient again, but I learned from the attending physician that the boy did well for several weeks, but that then he began to cough, to

expectorate and to tire easily. He was finally brought back to the hospital, where after roentgenograms showed definite changes in the right lung, the family otolaryngologist made a bronchoscopic examination. An uncracked pistachio nut was found in one of the right secondary bronchi, but the otolaryngologist was unable to remove it. The patient died several days later.

CONCLUSIONS

1. A foreign body which per se could not have passed the glottic chink, was found beyond the larynx, force having evidently accomplished the passage.

2. Early roentgenograms of a nonopaque, partially obstructing foreign body in a bronchus are of little aid to the clinician.

3. It is doubtful whether immediate bronchoscopic inspection would have been tenable in the peculiar circumstances of this case.

4. Preliminary use of the practice board often brings out unforeseen mechanical problems.

228 State Street.

Progress in Otolaryngology

Summaries of the Bibliographic Material Available in the Field of Otolaryngology

TONSILS AND ADENOIDS

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In reviewing the literature for 1932 on the tonsils and adenoids I have selected only those papers which promote discussion and those which, according to my judgment, show the advance made in this particular field of otolaryngology. An interesting paper by Boland shows the value of the Schilling blood count in cases in which complications develop following tonsillectomy. Gafafer's reports on the relation of adenoids and of tonsillectomy to the incidence of infections of the upper respiratory tract are particularly instructive. Two papers by Kelemen and Hasskó regarding the absorption of foreign matter by the tonsils represent further steps in the development of knowledge of the physiology of these organs. The reports of Waldapfel, Abt, Taptas, Hofer and others show that in the treatment of posttonsillitic pyemia ligation of the jugular vein has proved to be an important and necessary procedure in the management of this complication. Mayer's report of a large series of cases of peritonsillar abscess in which tonsillectomy was performed as a therapeutic measure is noteworthy. The communications of Hollender, Cohen and Balmer on the use of electrosurgical methods of tonsillectomy give an interesting review of the present status of the value of this method of surgical intervention.

GENERAL CONSIDERATIONS

Bradford[1] stated that in a group of 600 cases of scarlet fever a slight but statistically insignificant beneficial effect of tonsillectomy on the incidence of the disease occurred. The degree of severity of the disease was about the same in the tonsillectomized as in the nontonsillectomized patients. The average percentages of streptococci in the throat during the first few days of scarlet fever were about equal in the children in whom complications later developed and in those who

1. Bradford, W. L.: Tonsillectomy: Its Effect on the Incidence, Severity and Complications of Scarlet Fever and on the Number of Hemolytic Streptococci in the Throat, Am. J. Dis. Child. **44**:279 (Aug.) 1932.

escaped. This was true regardless of whether the child had previously been operated on. The rate of disappearance of the organism from the throat was about equal in children with and in those without tonsils. The tonsillectomized child, however, had a few less organisms throughout convalescence.

Boland [2] discussed briefly some of the literature on the effect of surgical intervention on the blood picture and reported his own study of the blood picture following tonsillectomy in 100 cases. The author's investigations were based on Schilling's hemogram table. Schilling differentiates between a degenerative and a regenerative nuclear shift. The regenerative shift is the one Arneth calls the shift to the left. The blood picture showed a leukocytosis which disappeared within three or four days. The shift to the left was always of slight degree. With the onset of a complication, the change in the blood picture always preceded the change in the clinical picture by one or two days. The severity of the complication was parallel to the regenerative nuclear shift. The author also mentioned that a lymphatic reaction of the blood was found before operation in most of the cases of chronic tonsillitis. He emphasized the importance of serial examinations and the fact that a change in the blood picture is to be evaluated only as one of many symptoms constituting the entire clinical picture.

In a report on the relationship of adenoids in adults to the incidence of the occurrence of common colds, Gafafer [3] made a study of 317 cases observed for thirty-five weeks from Sept. 29, 1929, to May 31, 1930. Of these adults, 235 had adenoids and 82 did not. The two groups showed no significant difference in respect to (*a*) frequency, (*b*) severity or (*c*) type of attack of disease of the upper respiratory tract (common cold).

Gafafer [4] made a further study of 275 adults from whom it was endeavored to secure reports of all infections of the upper respiratory tract (common cold). Of these adults, 138 had tonsils, and 137 had had the tonsils completely removed. The two groups presented no significant difference with respect to (*a*) frequency, (*b*) severity or (*c*) type of attack of disease of the upper respiratory tract (common cold).

Erdélyi [5] discussed tonsillectomy on the basis of his experience with 1,600 cases; he dealt especially with the precautions to be taken during

2. Boland, J.: Blood Picture Following Surgical Interventions, Especially Following Tonsillectomy, Ztschr. f. Hals-, Nasen- u. Ohrenh. **30**:339, 1932.

3. Gafafer, W. M.: Adenoids and Upper Respiratory Disease (Common Cold) in Adults, Ann. Otol., Rhin. & Laryng. **41**:517, 1932.

4. Gafafer, W. M.: Tonsillectomy and Upper Respiratory Disease (Common Cold) in Adults, Laryngoscope **42**:385, 1932.

5. Erdélyi, J.: Experiences with Sixteen Hundred Tonsillectomies, Monatschr. f. Ohrenh. **66**:596, 1932.

tonsillectomy, the complications, the beneficial results and the indications for intervention. The author experienced only 7 hemorrhages among 1,600 tonsillectomies. He attributed this mostly to careful hemostasis at the time of operation. He expressed the belief that waiting until the patient fully recovers from tonsillitis or from any complication of it and is in good general condition helps prevent complications following tonsillectomy. The indications and the various diseases resulting from tonsillitis are fully discussed.

Schmid [6] pointed out that it is generally known that tonsillitis may precede the first manifestation of tuberculosis. He gave several case reports, and in conclusion admitted that the relationship between tonsillitis and tuberculosis also has a reverse aspect: that chronic tonsillitis, by its association with an increase in temperature may simulate tuberculosis. But he expressed the opinion that because this fact is generally known by physicians, there may be danger that tuberculosis will be overlooked. He therefore emphasized once more that it should be kept in mind that tonsillitis may mask tuberculosis.

A study of the tonsils undertaken by Rhoads [7] was concerned with the pathologic changes in relation to the bacterial content and to the clinical condition of the patient. He expressed the belief that in many cases simple inspection reveals sufficient evidence for the removal of tonsils. In most cases, however, it is of little importance as compared with the evidence adduced by a careful history, ordinary laboratory examinations and a painstaking physical examination. Team work by the internist and the otolaryngologist is necessary. If general examination reveals a systemic disease that is usually associated with a focal infection and other more obvious foci are not discovered, the tonsils should be regarded as probable sources of infection regardless of their external appearance.

ANATOMY AND PHYSIOLOGY

Hoepke [8] made the statement that in order to understand the diseased tonsil one must first know the function of the normal tonsil. It must be decided as to whether the lymphoid nodules are to be considered germinal centers or reaction centers against penetrating inflammatory and toxic irritants. Hoepke made a comparative histologic study of normal lymphoid tissue in animals and in diseased tonsils immediately after tonsillectomy. On the basis of this investigation, he expressed the

6. Schmid, H. J.: Tonsillitis Masking Tuberculosis, Schweiz. med. Wchnschr. **62**:497, 1932.

7. Rhoads, P. S.: Pathologic Changes in the Tonsils: Their Relation to the Usual Indications for Tonsillectomy, Arch. Otolaryng. **15**:599 (April) 1932.

8. Hoepke, H.: Function of Healthy and Diseased Tonsils, Ztschr. f. Laryng., Rhin., Otol. **22**:1, 1932.

belief that the lymph nodules in the normal unirritated tonsil are germinal centers producing lymphocytes, but that when the tonsil is irritated, which occurs frequently because of its location, the germinal centers become reaction centers which decrease or discontinue the production of small lymphocytes to produce phagocytosing reticular cells. The nodules can be germinating or reaction centers, according to need. The tonsil becomes a menace only when it must be a reaction center too long. It requires a period of rest, a change from a reaction to a germinating center from time to time.

Kelemen and Hasskó [9] reported the first of a series of two experiments investigating the absorption of foreign material in the tonsils. The tonsils of young persons, which had been painted with a solution of arsphenamine a short time before their surgical removal, were examined after excision by Jancso's reduction test. Arsphenamine is conducted toward the lymphoid ground tissue only if the epithelium is intact. In the epithelium there were granular deposits in the protoplasm, especially in the nucleus, which has an elective receptivity for arsphenamine preparations. In the lymphoid ground tissue, storage of arsphenamine took place in the framework of the reticulum, the elements of which appeared as tubular structures with stained walls or as fibrils with incrustations. It is the cells which ensheath these fibrils or tubular fibers and which the authors call wall cells that constitute the actual storage elements of the reticulum. Blood and lymph vessels, especially the lymph spaces beneath the epithelium, take up the precipitate in the form of thrombi and also in their walls, but rapidly transport it to the underlying tissue. With the intravenous injection of massive doses of arsphenamine (which, however, can be made only in animals), the coarser elastic fibers of the parenchyma showed the strongest inhibition, thereby concealing the finer meshes of the reticular network. The region of the secondary nodules remains free from arsphenamine after the tonsils have been painted, as well as after intravenous injection, with the exception of occasional small thrombi in the vessels.

In a second series of experiments investigating the absorption of foreign matter by tonsils, Kelemen and Hasskó [10] employed colloidal gold, which was shown by Jancso to be a specific test medium for reticulo-endothelium. When a 1 per cent solution of colloidal gold was applied to the surface of the tonsils in human beings, it was found diffused within the superficial epithelium, but it did not penetrate any

9. Kelemen, G., and Hasskó, A.: Absorption of Foreign Matter in Tonsils: I. Experiments with Arsphenamine on Human Tonsils, Arch. f. Ohren-, Nasen- u. Kehlkopf. **130**:318, 1932.

10. Kelemen, G., and Hasskó, A.: Absorption of Foreign Matter in Tonsils: II. Tonsils and Colloidal Gold, Arch. f. Ohrenh-, Nasen- u. Kehlkopfh. **131**:222, 1932.

deeper. The intravenous administration of colloidal gold to guinea-pigs resulted in a heavy deposit of gold in the reticulo-endothelium of the spleen and liver, but no gold could be demonstrated histologically in the tonsils. There are no elements in the tonsils, in the walls of the blood vessels, along the basal reticulum or elsewhere capable of phagocytosing a specific test medium for reticulo-endothelium, such as colloidal gold. These experiments give no proof that there is any cellular system in the tonsillar tissue which can be ranked with the reticulo-endothelium of the spleen and liver.

PATHOLOGY AND BACTERIOLOGY

Waldapfel [11] reported his study of the pathologico-anatomic changes following nasal operations which prepare the way for the development of tonsillitis. Nasal tamponade and galvanocautery are the two operations most frequently followed by tonsillitis. The author first studied the changes that generally occur following these operations, and then studied the changes following these operations in cases later followed by tonsillitis. Bacteriologic investigation revealed that the bacterial flora, which is slight in the normal nose, is greatly increased by all types of tamponade. However, hemolytic streptococci in pure culture were obtained from the nose following tamponade or galvanocautery only in those patients in whom traumatic tonsillitis later developed. The pathologico-anatomic changes observed after light tamponade with white gauze were partial destruction of the superficial epithelium down to the basal membrane, hyperemia and leukocytic infiltration of the adenoids and the upper glandular layer. Iodoform gauze produced similar changes, but these were sometimes more extensive, consisting of small circumscribed areas of necrosis. In the cases of tonsillitis, the mucous membrane, following tamponade, was characterized by widespread destruction and necrosis extending into the erectile tissue layer and opening of the glandular and vascular lumen, which were frequently thrombosed. In this pathologic tissue, hemolytic streptococci were found in almost pure culture. Following nasal tamponade gram-positive streptococci were found in large numbers, within the follicles of tonsils that clinically were not diseased. The author expressed the belief that this invasion of the parenchyma by gram-positive streptococci, which has never been observed in a clinically healthy tonsil, but is known to be extensive in lacunar tonsillitis, might be considered as the last link in the chain leading from endonasal operation to the pathologic tonsillar changes occurring in tonsillitis. Waldapfel expressed the opinion that

11. Waldapfel, R.: Pathologico-Anatomic Factors in the Development of Traumatic Tonsillitis, Ztschr. f. Hals-, Nasen- u. Ohrenh. **30**:245, 1931.

there is a close connection between the nose and the tonsils in spite of the fact that experimental evidence is to the contrary.

In a study of the tonsils from the standpoint of irritative conditions of the crypts, Thiesbürger [12] pointed out that the essential cause of tonsillitis is an increase in the virulence of organisms usually present in these structures or a decrease of resistance of the patient. As a predisposing factor, stagnation of the cryptic contents is of primary importance in the development of the inflammation of the crypts. As probable factors that are responsible for the prolongation of the process, the author considered horny plugs, cornification and perhaps also tonsilloliths, but he did not consider nodules produced by Leptothrix responsible. Finally, he concluded that it is probable that the clinically manifest forms of tonsillitis begin with clinically latent acute inflammation of the crypts.

In a study of infection with Brucella abortus in man, Carpenter and Boak [13] isolated this organism from 8 of 56 pairs of tonsils. While the authors did not wish to convey the impression that infection with Brucella abortus is a cause of tonsillitis or of hypertrophied tonsils, nevertheless, in experimental and domesticated animals, the infection localizes in lymph and lymphoid tissue, frequently producing a focal or a general lymphadenitis, as well as a splenitis. At the onset of many cases of undulant fever there are reddening and injection of the fauces, pharynx, tonsils and peritonsillar tissue, not unlike those seen in an acute infection of the upper respiratory tract. Cervical adenitis is not unusual. That these pathologic changes are specific of infection with Brucella abortus is not known. The organism may invade the tonsil and multiply or accumulate there until the resistance of the host is decreased from fatigue or from disease, permitting its invasion of the blood stream.

In bacteriologic examinations of the tonsils Hofhauser [14] found, while searching for Actinomyces, organisms that resembled but were not the same as the true type. The tonsils were examined for fungi by both bacteriologic and histologic methods. Animal inoculation was also studied. The tonsils from 50 patients were used as the material for this study. A true colony of Actinomyces could not be found in a single instance, but in 15 of the 50 cases formations were found which resembled Actinomyces in form and size, although staining and con-

12. Thiesbürger, B.: Irritative Conditions of Crypts of Palatine Tonsils, Ztschr. f. Hals-, Nasen- u. Ohrenh. **28**:459, 1931.

13. Carpenter, C. M., and Boak, R. A.: The Isolation of Brucella Abortus from Tonsils, J. A. M. A. **99**:296 (July 23) 1932.

14. Hofhauser, J.: Occurrence of Actinomyces-Like Bodies in Palatine Tonsils, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **129**:293, 1931.

sideration of the finer structure aided in the differentiation from true Actinomyces. For this reason the author was not in favor of the term Actinomyces-like bodies, but recommended the designation Corpora lacunaria. He concluded that true Actinomyces which later cause actinomycosis occur rarely in the tonsils, but that Corpora lacunaria are found comparatively often.

In a study of infections of the throat with hemolytic streptococci and their relation to arthritis and arthralgia, Pilot [15] concluded that these diseases of the joints may complicate scarlet fever, erysipelas, wound infections and sore throat caused by these organisms. He showed that the complicating arthritis develops three or more weeks after the onset of the streptococcic disease, and that this suggests hypersensitiveness as an important factor. Epidemics of streptococcic sore throat may be responsible for a relatively small number of cases of acute and chronic arthritis of the so-called rheumatic type. He also remarked that the persistence of Streptococcus epidemicus in the tonsils is often associated with arthritis. Removal of the tonsils terminates the carrier state, and the associated arthritis frequently disappears.

Bézi [16] recorded the results of experimental tests of the action of saliva and extracts of tonsils and other organs on diphtheria toxin. Evidence is presented which seems to indicate that some specimens of saliva may exert an antibacterial action against diphtheria bacilli, and that saline extracts of tonsils and lymph nodes may inhibit the necrosing action of diphtheria toxin. It has not been determined whether or not these reactions are specific, and the experiments do not permit the drawing of definite conclusions.

The tonsils were carefully examined by Otto [17] in 45 patients who died of tuberculosis. In 74 per cent there were tuberculosis foci. In only 1 case was the infection presumably primary. Direct and indirect evidence makes it likely that the tonsils are usually infected by way of the blood stream. Histologic studies cannot disclose with certainty whether a tonsillar focus is due to an infection from the surface or to hematogenous propagation, particularly when the focus is advanced. Hematogenous development produces usually multiple, more or less uniform tubercles in the subepithelial and the perifollicular tissues. The follicles are rarely infected. A lymphogenous spread from the tonsils to the meninges was never observed.

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15. Pilot, I.: Infections of the Throat Due to Hemolytic Streptococci and Their Relation to Arthritis and Arthralgia, Arch. Otolaryng. **15**:71 (Jan.) 1932.

16. Bézi, I.: Study of Action of Saliva and Extract of Tonsils upon Diphtheria Bacillus and Diphtheria Toxin, J. Immunol. **22**:1, 1932.

17. Otto, J.: Tonsils and Tuberculosis, Beitr. z. Klin. d. Tuberk. **79**:187, 1932.

In a discussion of malignant disease of the tonsils, Ziegelman [18] stated that tumors of the lymphatic system are not uncommon. They may be benign or malignant. New growths and other pathologic lesions of these structures are characterized by the activity of such cells as lymphocytes and reticular and endothelial types. The author adopted the classification advocated by Ewing. The latter described lymphosarcoma of the tonsil as a true malignant neoplasm arising in lymphatic tissue from a proliferation of atypical lymphocytes. It occurs as a localized or a diffuse process, and develops from two particular types of cells, that is, the reticular cells and the lymphocytes. Differential diagnosis is not as simple as it seems. The history, the clinical aspect of the patient, the local appearance of the lesion in the tonsil and hematologic studies must be correlated to attain a satisfactory diagnosis. Ziegelman reported 2 cases of lymphosarcoma and 1 case of carcinoma of the tonsil.

COMPLICATIONS

Abt [19] stated that postanginal sepsis usually follows a tonsillar infection, or may be preceded by a pharyngeal phlegmon or an intratonsillar abscess. The pyemia may develop shortly after the primary focal infection, or the symptoms of sepsis may be deferred as long as four weeks after the primary disease, though an analysis of all cases shows that the average length of time that elapses is from ten to fourteen days after the original infection. This constitutes a latent period, during which the patient seems comparatively well. The occurrence of chills is of great importance, especially if they are observed after the angina has subsided. In all cases the author advocated prompt drainage of localized abscesses and ligation of the jugular vein in those cases in which symptoms of sepsis have occurred.

Taptas [20] discussed posttonsillitic pyemia, in regard to pathogenesis, diagnosis and therapy. He expressed the belief that in posttonsillitic pyemia the infection spreads along the lymph channels to the lymph nodes in the region of the jugular vein and from there by continuity to the latter. Frequently the veins become infected before the formation of a cervical abscess, and sometimes the veins become involved only after the formation of an abscess. At the onset of pyemia the tonsillar inflammation may have disappeared, but the infection in the cervical glands remains and the veins become infected from this focus. When a septic type of temperature develops, the author recommended ligation of the

18. Ziegelman, E. F., Malignant Disease of the Tonsil, with Special Reference to Lymphosarcoma, Arch. Otolaryng. **15**:697 (May) 1932.

19. Abt, I. A.: Postanginal Sepsis, J. Pediat. **1**:8, 1932.

20. Taptas, N.: Posttonsillitic Pyemia, Enucleation of the Tonsils During the Course of Peritonsillar Abscess and Acute Tonsillar Inflammations, Presse méd. **40**:414, 1932.

jugular vein. If a large peritonsillar abscess can be drained, this must be done promptly, and in cases of deep abscesses the tonsil should be removed.

Hofer [21] expressed the belief that surgical intervention in posttonsillitic septicemia should be undertaken in the earliest stage of propagation of the infection. In all cases in which this propagation is attended by chills, the first real chill should be considered an indication for intervention. Locally, a severe tonsillitis is usually followed by the formation of an abscess, which forms the basis for a septic condition. The large venous trunks of the neck may be sensitive to pressure, especially in cases of extensive thrombosis, but this sensitivity to pressure may be lacking as the disease may take its malignant course without extensive thrombosis of the large blood vessels, especially in the case of primary spread of infectious material into the blood stream. The author demonstrated by reporting 2 cases that good results can be achieved by ligation of the jugular vein and exclusion of the primary focus immediately after the onset of the first clinical manifestation of septicemia of tonsillar origin, even when no inflammation of the cervical tissues or the sheath of the vessels is demonstrable. Exclusion of the primary focus, whether still active or not, and interruption of the chief downward route of propagation, the jugular vein, are the first therapeutic requisites, but to be effective they must be undertaken in the first stage of the disease, after the first chill.

It is Linck's [22] opinion that the most satisfactory treatment of peritonsillar abscesses consists in performing a tonsillectomy as soon as the diagnosis of abscess is established. He considered simple incision inadequate not only from a prophylactic standpoint, but from a symptomatic standpoint as well. Tonsillectomy is preferred in the acute stage of peritonsillar infection because the abscess is more easily drained regardless of the location. Although puncture and incision may suffice to drain large bulging abscesses, those deeply situated are usually not drained by this method. After the tonsil has been removed, abscesses deep in the muscular bed can easily be drained. In 163 cases of uncomplicated peritonsillar abscesses in which primary tonsillectomy was performed in the acute stage in the author's clinic, there was not a single exacerbation of the condition due to the spread of infection. In conclusion, the author stated that systematic institution of tonsillectomy whenever a peritonsillar abscess is diagnosed or there is a suggestion of a spread of infection, whether it is in the early or the late stage, constitutes the foremost prophylaxis for complications, including the often fatal posttonsillitic septicemia.

21. Hofer, G.: Surgical Therapy of Posttonsillitic Septicemia, Monatschr. f. Ohrenh. **66**:587, 1932.

22. Linck, A.: Incision or Tonsillectomy in the Treatment of Peritonsillar Abscess? Arch. f. Ohrenh-, Nasen- u. Kehlkopfh. **131**:310, 1932.

Mayer [23] reported a case of circumscribed gravitation abscess originating in the peritonsillar region and descending into the mediastinum, which healed without surgical intervention other than incision of the peritonsillar abscess, from which the suppuration spread. In this case, eleven days after incision of the peritonsillar abscess the patient vomited a large amount of pus. Roentgenologic visualization of the abscess cavity by the injection of an opaque fluid showed that the suppuration descended into the spatium retropharyngeum and the posterior portion of the mediastinum between the esophagus and the spinal column, ending in the thorax at the level of the fourth thoracic vertebra. The patient recovered under conservative therapy. The necessity of differentiating between a circumscribed gravitation abscess and the much more frequent phlegmons in the throat with general septic symptoms is stressed.

Murphy [24] reported 2 cases in which a peritonsillar abscess followed the injection of fluid into the peritonsillar space for the purpose of anesthesia. In the first case the abscess developed because injections were made into both tonsils, and on account of the condition of the patient only one tonsil was removed. In the other case injections had been made into the peritonsillar space previous to the use of electrocoagulation, and eight days later an abscess developed. The author referred to 3 other cases in which he knew there had been similar occurrences. The conclusion is made that the occurrence of a peritonsillar abscess following the injection of fluid into the peritonsillar space is a hazard to be considered. Injection into one tonsil at a time is recommended in performing a tonsillectomy under local anesthesia.

According to the observations of Horning and Wenger,[25] patients of all ages with unilateral quinsy have a homolateral dilatation of the pupil. The sign is clearer when the distention from the abscess is greater. The authors have had the experience of draining the abscess cavity and seeing the mydriasis disappear, and when iodized poppy seed oil was injected into the abscess cavity the sign reappeared. A roentgenogram showed the abscess cavity to be water-tight, with complete closure toward the tissues of the neck. The mydriasis again disappeared when the cavity was emptied. The mechanism of its production is either by direct irritation of the sympathetic system or by a "repercussivity" of bulbar origin.

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23. Mayer, W.: Posttonsillitis Gravitation Abscesses, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **131**:125, 1932.

24. Murphy, A. B.: Peritonsillar Abscess Following Injection of Fluid into the Peritonsillar Space, J. A. M. A. **99**:1945 (Dec. 3) 1932.

25. Horning, T., and Wenger, S.: Peritonsillar Abscess and Mydriasis, Rev. d'oto-neuro-opht. **9**:580, 1931.

In their clinical experience Combiesco and his associates [26] have found that the treatment of tonsillar abscesses with staphylococcus bacteriophage assures a rapid cure, usually within a few days. This treatment may be employed at any stage in the evolution of the abscess, but is most indicated at the stage of infiltration. Treatment of the abscesses with antistaphylococus or antistreptococcus filtrate prepared according to Besredka's method, as well as sterile peptone broth and physiologic solution of sodium chloride, gave the same results. The authors concluded that the effect of injection of these substances is nonspecific, and that the action consists of a stimulation of the defense elements of the organism.

Frank [27] reviewed the literature on the subject of pulmonary abscess following tonsillectomy, and reported a case from his own experience in which inhalations of carbon dioxide were employed as a therapeutic agent. After a hemorrhage on the second day following tonsillectomy, the beginning symptoms of an abscess of the lung developed. Inhalations of carbon dioxide, through its action on respiration, caused the patient to cough up a large clot, which if it had remained in the lung would have caused an abscess. The author recommended the use of inhalations of carbon dioxide in similar cases.

Halpern [28] reported a group of 6 cases of encephalitis occurring as a complication of tonsillitis. Two hypotheses are offered as to the etiology: (1) There may be a causal relationship between the present increasing number of cases of encephalitis and the epidemic of tonsillitis, and (2) there exists an epidemic of encephalitis due to an unknown virus which seems to have a special affinity for the brain stem and needs another factor to activate it, and another infection such as an angina may be the activating factor in a person who had previously been resistant to the virus.

Houser [29] reported a case in which death occurred eleven hours after tonsillectomy from the local application of cocaine. For the operation on the tonsils a 1 per cent solution of procaine hydrochloride was used without any noticeable effects. Eleven hours after the operation, during the control of a postoperative hemorrhage, a solution of 10 per cent cocaine was applied locally to the tonsillar fossa. Within a few minutes a generalized convulsion occurred, followed by the other symptoms of cocaine poisoning. The patient died in spite of all therapeutic measures.

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26. Combiesco, D., Tzetzu, I., and Popesco, M. S.: Mechanism of Action of Bacteriophage in Treatment of Tonsillar Phlegmons, Presse méd. **40**:1386, 1932.

27. Frank, I.: Pulmonary Abscess Following Tonsillectomy, Ann. Otol., Rhin. & Laryng. **41**:550, 1932.

28. Halpern, F.: The Frequent Appearance of Encephalitis After Nonspecific Tonsillitis, Jahrb. f. Psychiat. u. Neurol. **48**:154, 1932.

29. Houser, K. M.: Sudden Death Eleven Hours After Tonsillectomy Following Local Application of Cocaine, Arch. Otolaryng. **15**:291 (Feb.) 1932.

This case is reported as a wraning against the use of cocaine in the throat with surgical procedures.

Skoog [30] reviewed the literature on the subject of spontaneous hemorrhages of the tonsillar regions and divided the cases into three groups, namely: (1) hemorrhages due to septic processes in the tonsils and adjacent tissues, (2) hemorrhages associated with localized ulceration of the tonsils and (3) truly spontaneous hemorrhages. In the cases in group 1 he recommended ligation of the external carotid artery. Ligation of the common carotid is considered hazardous and should be performed only in case of necessity. In the second group of cases, in which life is not directly threatened, Skoog said that general therapy is sufficient. In the third group of cases, or the true spontaneous hemorrhages, the author advised care in differentiation from gastric and pulmonary hemorrhages.

TREATMENT

A paper by Hollender [31] deals with the fallacies as well as the possibilities of electrosurgical tonsillectomy. After a discussion of the current methods in use, and the advantages and disadvantages of electrosurgical methods, the author concluded that while diathermy for removing the tonsils has gained favor and has become the method of choice of some specialists, its use is not to be recommended as a substitute for surgical procedures in the majority of cases. There are, however, definite advantages in an adequate substitute method when surgical intervention is considered hazardous in the presence of certain systemic diseases. Since electrocoagulation of the tonsils is a scientifically sound method for selected cases, the specialist should familiarize himself with the technic of its use.

After four years' experience in the use of electrosurgery, Cohen [32] stated that it has a definite place as a valuable adjunct to surgical intervention, but he expressed the belief that the use of this method should be applied to the tonsils only in certain selected cases. Electrocoagulation of the tonsils is especially valuable in aged persons, in the presence of tuberculosis, organic heart disease, nephritis or hypertension and in any condition in which surgical methods are contraindicated. The absence of shock, the slight pain and reaction following its use and its economic advantages are factors highly in its favor.

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30. Skoog, T.: Spontaneous Hemorrhages of Tonsillar Regions, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **130**:206, 1932.

31. Hollender, A. R.: Electrosurgical Tonsillectomy: Advantages and Limitations of the Fractional Electrocoagulation Method, Ann. Otol., Rhin. & Laryng. **41**:216, 1932.

32. Cohen, L.: Electrosurgery as an Aid in Rhinopharyngology, Ann. Otol., Rhin. & Laryng. **41**:329, 1932.

In the employment of electrosurgery Balmer [33] stressed the necessity of a thorough knowledge of the various currents used. He described in detail the various currents with their therapeutic action and indications. He also described a method of extirpation of the tonsils by the use of electrosurgery. Electrocoagulation is not recommended to replace surgical removal of the tonsils, but it is better suited in certain selected cases. It is a method par excellence for the removal of stubs of tonsils and tissue adhesions. Balmer further stated that the surgeon should be equipped to use the method best suited to the conditions at hand, and should not be limited by a lack of knowledge, ability or equipment or by prejudice.

Smith [34] has devised an instrument which is used according to the Sluder technic. It has a snarelike arrangement instead of the solid metal loop of the Sluder instrument. The hemostasis is excellent, because the tonsil is crushed instead of cut.

For the control of pain following tonsillectomy, Laszlo [35] has found through his observations that tablets containing ethylaminobenzoate, which are dissolved in the mouth, are ideal for this purpose. They may also be used in esophagoscopy.

On the basis of his observations on the use of the modified Rose position for tonsillectomy under general anesthesia, Ziegelman [36] concluded that the danger of aspiration with its complications is practically eliminated. This position is ideal for orientation of the operation, and hemorrhage can be easily controlled.

A study of local anesthesia for tonsillectomy was undertaken by Trotter [37] to determine whether the arrangement of the sensory nerves entering the tonsil was such that a method of "nerve block" could be employed. It was thought that such a method would reduce the number of needle punctures and the large amount of solution usually required. First, the anatomy of the tonsillar nerves is considered. By the use of methylene blue (methylthionine chloride, U. S. P.) in the anesthetizing solution, the location of the deposition of the dye was noted in the tonsillar fossa. Trotter used a solution of 1 per cent quinine and urea hydrochloride in the amount of 2.5 cc. to each tonsil.

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33. Balmer, F. B.: Electrosurgery and the Otolaryngologist: Including Extirpation of the Tonsils by Electrocoagulation and Diathermocryptectomy, Arch. Otolaryng. **15**:503 (April) 1932.

34. Smith, U. S.: Tonsillectomy with a Simple Snare; Sluder Technic; Minimum Hemorrhage, Arch. Otolaryng. **16**:92 (July) 1932.

35. Laszlo, A. F.: Control of Pain Following Tonsillectomy, Laryngoscope **42**:66, 1932.

36. Ziegelman, E. F.: Tonsillectomy Under General Anesthesia in the Modified Rose Position, Laryngoscope **42**:49, 1932.

37. Trotter, H. A.: Local Anesthesia in Tonsillectomy, Arch. Otolaryng. **15**: 435 (March) 1932.

Ten minutes are allowed for full anesthesia. He used this nerve block anesthesia in 500 cases with satisfactory results. At the time of operation, the anesthesia is excellent, and the recovery of the patient is much smoother because the anesthesia lasts for several days.

Bernfeld [38] expressed the belief that one of the chief causes for the frequent failure of operations on the adenoids is the lack of consideration given to the various morphologic peculiarities of the epipharynx. He emphasized the importance of an accurate examination of the nasopharynx prior to possible operation, and discussed the three most important methods of examination, namely, postrhinoscopic mirror examination, palpation and roentgenographic examination. The importance of diagnosing an infantile nasopharynx, which can lead to a number of disturbances in the region of the ear and pharynx, is stressed. He pointed out the existence of asymmetries of the pharynx and epipharynx hitherto unnoticed. The choice of curet must be exactly suited to the morphologic conditions of the nasopharynx, whether the operation is performed blindly or under direct visual control. The indication and prognosis of nasal disturbances in the presence of adenoids can be estimated correctly only if the relation between the structure of the nasopharynx and other obstructions of the nasopharynx have been ascertained with certainty.

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38. Bernfeld, K.: Causes of Failure in Adenoid Operation, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **131**:234, 1932.

Abstracts from Current Literature

Ear

THE FUNCTION OF THE BRAIN IN AUDITION. L. E. WILEY, J. Comp. Neurol. **54**:109 (Feb.) 1932.

This study was undertaken to determine what part (or parts) of the cerebral cortex is used in the formation and retention of an auditory habit and also to gain some knowledge of the functioning of the auditory mechanism. Rats were first trained to an auditory stimulus and then subjected to cortical operation. The lesions varied in locus and extent, covering practically the entire neopallium and involving from 2.6 per cent to 31.7 per cent of the total neopallial cortex. Ten days after operation, the rats were returned to the problem and retrained until they again reached the original criterion of learning. They were then killed; the brains were removed and sections were made to determine the place and extent of the lesion.

The results show that Fortuyn's area *p*, which includes the posterolateral part of the cerebral cortex, delimits the auditory mechanism fairly closely. Large destruction of area *p* may abolish an auditory habit, but the habit may be reacquired. The data show that there is a relationship between the extent of the destruction and the degree of postoperative amnesia.

FRASER, Philadelphia. [ARCH. NEUROL. & PSYCHIAT.]

OTITIC HYDROCEPHALUS: REPORT OF THREE CASES. C. P. SYMONDS, Brit. M. J. **1**:53 (Jan. 9) 1932.

In otitic hydrocephalus up to a certain point clinical evidence suggests a cerebellar abscess secondary to otitis media, but the cases reported were felt to be due to hydrocephalus without previous meningitis, extradural abscess or lateral sinus thrombosis. There appeared to be merely an excessive amount of spinal fluid. The most striking feature of this condition is the swelling of the optic disks. This with hemorrhage and exudate frequently amounts to several diopters. In cerebral or cerebellar abscess, on the other hand, the optic disks commonly show no more than a slight venous engorgement. The degree of swelling as met with in hydrocephalus is rarely seen in cases of abscess, and is then accompanied by extreme drowsiness and malaise.

In striking contrast, however, to the appearance of the fundi is the general condition of the patient. Between the attacks of headache and vomiting he is likely to be alert, lively and of good appetite. The patient with an abscess, on the other hand, generally looks and feels ill and is dull, constipated and without appetite. Lateral sinus thrombosis, however, associated with otitic hydrocephalus, complicates the picture.

In cerebellar abscess localizing signs, most notably nystagmus toward the side of the abscess and incoordination in the use of the upper limb on the same side, are characteristic, as the common situation for abscesses is temporal and cerebellar. "Word forgetfulness" is almost always found with a left temporal abscess, even when such an abscess occurs in a right-handed person. A right temporal abscess, however, is relatively silent, and the differential diagnosis from otitic hydrocephalus is more difficult.

Analysis of the spinal fluid gives the final diagnosis. In otitic hydrocephalus there are high pressure and normal fluid, that is, no excess of cells or protein.

The treatment is by lumbar puncture. Enough fluid should be removed at each puncture to bring the pressure to normal, and this should be repeated as

long as there is evidence of increased pressure. Three cases are reported. In these cases, however, there was complicating lateral sinus thrombosis without any actual infectious process of the membranes.

ROYSTER, University, Va. [AM. J. DIS. CHILD.]

TECHNIC OF VESTIBULAR EXAMINATIONS IN NEUROLOGY. J. RAMADIER and R. CAUSSÉ, Rev. d'oto-neuro-opht. **10**:305 (May) 1932.

Examination of the cochlear apparatus is not included in this discussion for the reason that rarely, except in lesions of the cerebropontile regions, does an intracranial disease cause auditory troubles. In diseases of the internal ear a caloric test to determine whether the labyrinth is excitable, hypo-excitable or inexcitable is sufficient; but in intracranial disturbances of the central vestibular pathways the examination must be complete. In the former the symptomatology is always essentially the same; in the latter the vestibular manifestations exhibit a great many modifications. It is true that the symptomatology of otolabyrinthine conditions presents variations, but they are quantitative; the phenomena, their order and even their evolution are of a constant type. Almost always it embraces the three fundamental phenomena: vertigo, nystagmus and spontaneous deviations of the trunk and limbs. The spontaneous nystagmus is always horizontal-rotatory, and the spontaneous deviations are bilateral and involve the various segments of the body. In the same way, alterations of reflectivity concern all the fundamental reactions—vertigo, nystagmus and deviations. These alterations are found with all three classic vestibular tests. In addition, this syndrome is usually accompanied by auditory disturbances. Each of the deviations of the trunk and extremities is made toward the same side, and the nystagmus is toward the side opposite the deviations. When there is total areflectivity to the tests, the spontaneous signs of irritation are not long in disappearing.

On the other hand, the symptomatology in intracranial conditions of the vestibular pathways is characterized by extreme variations and numerous modifications of the composition of the syndrome, its arrangement and its evolution. Frequently it is incomplete, often being represented by only one sign, spontaneous nystagmus or deviation of the arms, and even this sign may be manifested only in part. The nystagmus may be horizontal, rotary, vertical or oblique; the deviation, unilateral or limited to one member or a segment of one member. This incomplete character is found also in the provoked reactions. One reaction may be absent while the others are present, such as nystagmus without vertigo or the reverse, deviation without nystagmus; or rotary nystagmus may not occur while horizontal nystagmus is normal, or the deviation may be limited to one limb. Again, the reaction to one test may be exaggerated and that to another diminished; the spontaneous inclination of the body in the Romberg test may be in an inverse direction to the deviation of the arm; spontaneous nystagmus and spontaneous deviation may be toward the same side, or the spontaneous nystagmus may be bilateral. Finally, the evolution of the phenomena is not subject to rule: A violent nystagmus may persist indefinitely, while tests indicate a total areflectivity of the labyrinth. Any departure from the typical vestibular syndrome suggests the probability of an endocranial lesion.

The details of the examination are discussed under the following heads: inspection of the tympanic membrane; spontaneous reactions (vertigo, nystagmus, segmental deviations, falling [the Romberg test] and walking with blinded eyes); provoked reactions, and the caloric galvanic and rotation tests.

The presence of impacted cerumen or a perforated drum membrane will influence the results of the tests. Spontaneous vestibular reactions are subjective (vertigo) and objective (modifications of muscular tonus, shown in the eyes, limbs and body musculature). True labyrinthine vertigo is accompanied by a sensation of displacement of the body and the erroneous sensation of rotatory displacement of surrounding objects. The patient must differentiate it from headache, giddiness and the tendency to lipothymia, which may simulate it. A true rotary vertigo is rare in patients with neurologic conditions. Except in acute exacerbations of

disseminated sclerosis, syringobulbia, Friedreich's disease and, perhaps, certain tumors of the fourth ventricle, vertigo in neurologic disease is a little intense and is out of proportion to the other spontaneous signs. The absence of vertigo in the presence of an intense spontaneous nystagmus is presumptive evidence of a central disturbance. Spontaneous nystagmus is searched for in the four directions of gaze and in the act of accommodation, with and without the use of Bartel's spectacles; the influence of the different positions of the head on the nystagmus is observed; its intensity, form and direction are noted. Precise observations are necessary.

To determine segmental deviations, the test of the extended arms is preferred. (Static tests give more constant and trustworthy results than kinetic.) The deviation of the arms occurs only after a short interval and rarely attains a large amplitude. It is one of the most sensitive and precise tests of all. Segmental deviations almost always consist of movements of abduction and adduction. The Romberg test is best made with the use of a plumb line, and it should be noted whether changes in the position of the head influence the inclination. The pelvic girdle test is, in effect, the pulsion test of Marie and Bouttier. In executing the test of walking blindly, the observer follows the patient, retaining the same position in relation to him when giving the commands.

Instrumental tests are antiphysiologic because of their intensity and their nature, and are disagreeable for the patient. In executing the caloric test two methods are used, one weak and one strong. In the former, the ear is filled with water at 25 C. After thirty seconds the head is placed upright, Bartel's spectacles are put on the patient, and the eyes are examined for nystagmus; the head is put backward 60 degrees, in which position a horizontal nystagmus should appear; then the head is inclined 45 degrees toward the shoulder opposite the ear that is being tested, in order to bring out rotatory nystagmus. It should be noted that the rotatory nystagmus does not always appear even in normal persons. To investigate rotatory nystagmus properly, all three tests, douching, rotation and galvanic, are required. The head is now placed upright, the test for deviation of the extended arms is made, and the patient is questioned in regard to vertigo. If the weak test is inconclusive, resort is had to the strong test, which consists in irrigating the ear with water at 25 C., using 100 cc. or more. The caloric test gives information as to the degree of excitability of each labyrinth and as to the presence of each form of nystagmus, horizontal and rotatory. In a normal person, nystagmus lasting about one minute, deviation of the arms and vertigo should appear after the weak test.

The galvanic test reveals the threshold of excitability to the current for nystagmus and falling, and the form of nystagmus, which is normally horizontal-rotatory. The test is applied with the patient standing; the current is gradually applied until swaying begins (at from 2 to 3 milliamperes) and nystagmus appears (3 to 4 milliamperes). If by douching or rotation, rotatory nystagmus has not been elicited, the galvanic nystagmus will be horizontal. Bourguignon's studies of chronaxia should be extended.

The usual technic of ten turns in twenty seconds is employed in the rotation test. The past pointing reaction is not utilized because of its unreliability. The optimum position for eliciting rotatory nystagmus is with the patient on a turning table with the head 90 degrees backward from the vertical. To elicit vertical nystagmus the head of the patient is turned to the right and the table rotated. The reaction of vertical nystagmus is remarkably stable in contrast to rotatory nystagmus. Horizontal nystagmus is elicited by turning the patient with the head upright and well supported.

Rotation furnished important information. Postrotatory nystagmus is the result of two excitations, peripheral and central; the former is due to movement of the endolymph, the latter to a brusk interruption of the reflex caused by the rotation (per-rotatory). The physiologic response to rotation is the per-rotatory nystagmus. Even turning of the head through an arc of 90 degrees is sufficient to produce nystagmus, but it is ordinarily masked by fixation of the eyes. It may be perceived by palpation through the closed lids.

DENNIS, Colorado Springs, Colo. [ARCH. NEUROL. & PSYCHIAT.]

RARE COMPLICATIONS OF SCARLET FEVER. B. S. HIRSCHBERG, Jahrb. f. Kinderh. **136**:243 (Aug.) 1932.

In a case of mild scarlet fever, on the sixth day streptococcic peritonitis developed and on the seventh day death occurred. Autopsy revealed no other complications.

In a case of moderately severe scarlet fever, complicated by bilateral otitis media and later by purulent ethmoiditis, both complicating conditions apparently subsided. On the fifty-fourth day, headache and vomiting occurred, and on the fifty-sixth day coma. The neck was stiff; Kernig's and Trousseau's signs were positive, and the pupils were sluggish. Death occurred. Autopsy revealed meningitis over the base of the frontal lobes and over portions of the cerebellum, an abscess under the corpus caudatum and pus in the lateral ventricle. Streptococci were present.

In case 3, death occurred on the thirtieth day, following signs of meningitis. Autopsy showed acute nephritis and several cerebral abscesses.

In case 4 (observed by Dr. Awdejeff) the onset of scarlet fever was severe. There were bilateral otitis media and long-continued fever. Death occurred suddenly on the fiftieth day. Section revealed rupture of the aorta 6 cm. beyond the valves, due to abscess in the wall of the vessel.

CLAUSEN, Rochester, N. Y. [AM. J. DIS. CHILD.]

NOISE INJURY THROUGH BONE CONDUCTION. B. LANGENBECK, Ztschr. f. Hals-, Nasen- u. Ohrenh. **30**:609 (July 22) 1932.

Langenbeck reports experiments aimed to throw light on the question as to whether the sound injuries occurring in employees of noisy industries result from air sounds or from ground sounds. By air sounds he means the sound vibrations of the air which are transmitted to the labyrinth directly by way of the tympanum and ossicles (air conduction) or through the skull (bone conduction). By ground sounds he means vibrations of solid objects which are transmitted by way of the ground to the feet and conducted by way of the skeletal bones and the base of the skull to the labyrinth (body conduction). Since every vibration of the ground also produces air vibrations, ground sounds are perceived by way of air conduction as well as through body conduction. The author measured the hearing capacity for ground sounds through body conduction alone by means of a bone telephone applied directly to the feet. To measure the hearing capacity for the same ground sounds by way of air conduction alone, a pasteboard membrane was connected with the bone telephone, which was then removed from contact with the feet and placed at body length from the ear. It was found that ground sounds are perceived much better by way of the air sounds produced through the ground vibrations than by way of body conduction. Low tones were heard at least ten times as well by air conduction as by body conduction, and for high tones this ratio was increased. In persons with normal hearing, sound injury by body conduction does not need to be practically considered, because it is necessarily accompanied by an injury through air sounds, which, in all tone ranges coming into consideration, is at least ten times as strong, and is probably over a hundred times as strong. Special precautions for the protection of employees against ground sounds, such as felt shoes, are therefore useless and should be replaced by protection against air sounds.

EDITOR'S ABSTRACT.

INJURY OF ACOUSTIC NERVE BY CARBON MONOXIDE: CASE. T. HÜNERMANN, Ztschr. f. Laryng., Rhin., Otol. (Teil 1: Folia oto-laryng.) **22**:418 (June) 1932.

Hünermann reports a rare case of direct injury of the hearing and of the apparatus for equilibrium by inspiration of carbon monoxide. The hearing disturbance consisted in a temporary bilateral bradyecoia which disappeared almost completely within three months. The middle ear was unaffected. Static ataxia dominated the clinical picture. The vestibular response to caloric stimuli was

decreased. Spastic symptoms were found on examination. One physician attributed the symptoms to cerebellar injury, whereas another physician attributed them chiefly to a tendency to psychogenic reactions with possible slight organic disturbances. The disturbances in equilibrium persisted until death from coronary disease about a year after the inspiration of the carbon monoxide. Necropsy revealed several lesions in the cerebellum, consisting in glial scars under the gray matter of the cortex with numerous amylaceous bodies and a few globules of fat. There were dilatations of the vessels in these regions and slight perivascular cellular infiltrations. As there were no significant signs of arterial disease in the region of the brain, the lesions were attributed to the carbon monoxide. The author thinks that the hearing disturbances were also of central origin and resulted from hemorrhage. The extravasations were small and did not lead to permanent injury of the brain. The absence of any signs of injury of the labyrinth and the bilaterality of the disturbance support this view. This case shows the difficulty of distinguishing psychogenic from organic disturbances, and that objectively demonstrable vestibular disturbances and especially differences in excitability should be considered important signs of an organic vestibular injury. It shows further that carbon monoxide poisoning must be considered as one of the many causes of sudden deafness or bradyecoia occurring with or without disturbances in equilibrium.

EDITOR'S ABSTRACT.

ELIMINATION OF FIXATION IN OBSERVATION OF NYSTAGMUS. H. KOBRAK, Ztschr. f. Laryng., Rhin., Otol. (Teil 1: Folia oto-laryng.) **22**:426 (June) 1932.

Kobrak describes a method of eliminating fixation in the observation of nystagmus which meets the following requirements for a satisfactory observation: (1) The optic contact with the surroundings and the influence of light must be eliminated; (2) the observation of the bulbus movements must be unhindered, and (3) the method must be easy and applicable anywhere. The method consists in covering one eye with a shield and placing a black opaque glass cap on the other eyeball, which follows the movements of the bulbus. On the convex side of the eyeball cap is a small peg with a white ball mounted on its free end. This facilitates placing the cap, and its free end, acting as a lever, amplifies the nystagmus beat. The patient is in the conditions offered by a dark room, but the physician is not impeded by the disadvantages of a dark room. Fixation is absolutely eliminated. In patients who are bothered by frequent blinking of the lids, this cap (due to the peg) permits the detection of nystagmus beats, which could not be seen if glasses were employed. It is equally applicable for caloric and rotatory tests, and especially facilitates the intrarotatory test. The technic of adjustment is easy. The conjunctival sac is anesthetized with 1 or 2 drops of 2 per cent cocaine a short while before the cap is fitted. Experiment showed that use of the correct size of cap prevents the possibility of the eyeball moving independently of the cap, and also that there is no untoward mechanical influence on the motion of the eyeball.

EDITOR'S ABSTRACT.

Pharynx

ABSORPTION OF FOREIGN MATTER IN TONSILS: TONSILS AND COLLOIDAL SILVER. G. KELEMEN and A. HASSKÓ, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **132**:326 (Sept. 21) 1932.

Kelemen and Hasskó's study of the absorption of colloidal silver in the tonsils constitutes the third series of experiments in an investigation of the absorption of foreign matter in the tonsils. Reports on the first and second series of experiments, dealing with the absorption of arsphenamine and of colloidal gold, appeared in the *Archiv für Ohren-, Nasen- und Kehlkopfheilkunde* (**130**:318 [Feb.] 1932; **131**:222 [May] 1932) and were abstracted in the ARCHIVES OF OTOLARYGOLOGY (**16**:587 [Oct.] 1932). In the present series, the authors experimented with collargol and electrocollargol on human beings and rabbits. With one exception, no silver

could be histologically demonstrated within the tonsillar tissue after application of collargol to the surface of human tonsils; neither could silver be demonstrated in the tonsils after intravenous injections of customary doses of electrocollargol. Collargol and electrocollargol injected intravenously into rabbits in the doses they tolerated did not reach the tonsils; only when lethal doses were injected could traces of silver in the tonsils be demonstrated. With injection of collargol after elimination of the reticulo-endothelium of the spleen and liver by colloidal copper (amounts up to the lethal dose), the tonsils also remained free from silver even when conditions for phagocytosis were improved by gelatin. Likewise, silver injected into animals after splenectomy and blockade of the liver by copper, and silver injected into a human being after splenectomy, could not be demonstrated in the tonsils of the experimental subjects. The authors conclude that the tonsil does not possess any elements capable of absorbing significant amounts of silver. Even in severe generalized argyrosis there is no significant absorption of silver in the tonsils as there is in the tissue of the surrounding organs.

EDITOR'S ABSTRACT.

FUNCTION OF THE TONSILS. H. SCHERF, Virchows Arch. f. path. Anat. **283**:74, 1932.

Full-grown rabbits were used in an investigation of the function of the tonsils. When colloidal trypan blue or silver or finely particulate india ink was painted on the surface of the tonsils, the material was not taken up by the lymphoid tissue. Spraying of the same substances into the pharynx led to deposition of the material on the surface epithelium, but it was not transferred to the tonsil itself. The surface of the tonsil may mechanically remove foreign particulate matter from inspired air. When the experimental materials were injected into the submucous tissues of the nasopharynx and mouth, they were stored in macrophages of the peritonsillar tissue, but did not reach the tonsillar tissue itself, indicating that peritonsillar inflammatory processes may result from lymphatic transport from the submucous tissues. When the materials were injected into the common carotid artery, the material was stored in the capillary endothelium, from which it was passed to the surrounding tissue. This process was most marked in the subepithelial capillaries, the material passing to the surface of the tonsil and being excreted. The tonsils may apparently function in the removal of foreign material from the blood stream.

SCHULTZ, Evanston, Ill. [ARCH. PATH.]

BLOOD PRESSURE-REDUCING EFFECT OF TONSIL EXTRACT. C. BISCHOFF and K. WEINAND, Ztschr. f. Hals-, Nasen- u. Ohrenh. **30**:647 (July 22) 1932.

Bischoff and Weinand report the chemical extraction of acetylcholine from human tonsils. The extract was found to have a strong reducing effect on the blood pressure of cats. Since Bischoff, Grab and Kapfhammer have shown that acetylcholine is contained in many other organs, the authors think that the mere presence of this blood pressure-reducing substance in the tonsils does not justify designating the latter as glands of internal secretion.

EDITOR'S ABSTRACT.

SEPTICEMIA FOLLOWING TONSILLITIS. H. CLAUS, Ztschr. f. Laryng., Rhin., Otol. (Teil 1: Folia oto-laryng.) **23**:58 (Sept.) 1932.

Claus discusses septicemia following tonsillitis, particularly in regard to diagnosis and therapy. After blood has been taken for bacteriologic and cytologic examinations, a careful anamnesis obtained and examination of the larynx and pharynx completed, the exterior of the neck should be carefully examined. Sensitivity to pressure in the angle of the jaw is significant if there are no acute manifestations in the tonsils. Cord formations are found in phlegmons and glandular infection, and are not a septic symptom unless the blood vessels or the mediastinum is involved. Careful study of the general condition is valuable. Special examina-

tion of the lungs, heart and kidney to determine metastases and of the eyes for symptoms of cavernous sinus thrombosis should be made. Thrombophlebitis of the jugular vein toward the sigmoid sinus may occur. Exposure of the sinus and removal of the thrombus may save the life of the patient. The author thinks that there is always a phlegmonous process in the loose connective tissue of the parapharyngeal space, which spreads to the near or distant lymph vessels or, more often, to the veins, and that the infection is never spread directly via the lymph or blood vessels. The phlegmon spreads toward the mediastinum more often than toward the cranial cavity. Surgical intervention for inspection of the large vessels should be undertaken whenever possible and should consist in an incision along the inner edge of the sternocleidomastoid muscle from the tip of the mastoid process to the clavicle. If the infiltration is severe and orientation is difficult, cervical mediastinotomy is indicated to find the veins; they may then be traced upward to the area of infiltration. Diseased vessels should be resected, whenever possible, following ligation. The healthy jugular vein should be ligated when the facial vein is diseased and in doubtful cases. Elimination of the actual septic focus is important. The author supplements control of the blood vessels with tonsillectomy, since a focus of suppuration is often found behind the normal appearing tonsil. Enucleation of an acutely inflamed tonsil need not be shunned if the interstitial spaces of the connective tissue have been previously exposed from the exterior and no injection is made into the infected area. Metastases in the lung are not a contraindication to intervention.

EDITOR'S ABSTRACT.

TONSILLECTOMY IN INTERNAL DISEASES. J. HORÁK and L. POLEDUÁK, Ztschr. f. Laryng., Rhin., Otol. (Teil 1: Folia oto-laryng.) **23**:135 (Sept.) 1932.

Horák and Poleduák discuss the results of tonsillectomy in internal disease on the basis of personal observations gathered since 1924. Tonsillectomy was usually performed after regression of the acute tonsillitis and the acute attack of internal disease, except in cases in which the acute attack lasted weeks and months without exhibiting any tendency to improve. In cases of sepsis, endocarditis and nephritis tonsillectomy was undertaken as soon as a relation between the internal disease and the tonsillar disease was ascertained. In cases of chronic disease, tonsillectomy was performed at any stage during a subfebrile period. The best results were obtained in cases of articular rheumatism. Only a small percentage of patients were treated by tonsillectomy, but of those so treated, 75 per cent were cured, 23 per cent were improved, and only 2 per cent were not improved. In cases of heart disease the results were also good, but 13 per cent of the patients showed no improvement. Among the cases of renal disease in which tonsillectomy was performed, focal nephritis occurred most frequently and responded most favorably. Good results were also obtained in cases of sepsis of oral origin. Among the patients with neurologic disturbances who were treated by tonsillectomy, the best response was obtained in those with chorea minor. Comparison of the results obtained by tonsillectomy in internal diseases with those obtained by other forms of treatment shows the therapeutic value of tonsillectomy in internal diseases of a generally septic character. The authors list the contraindications. They point out that if the hematogenous spread of infectious material from the tonsils into the body has been going on a long time, tonsillectomy often cannot arrest the septic process in the body.

EDITOR'S ABSTRACT.

RARE CASE OF MUCOCELE OF ETHMOID CELLS. C. MORITA, Monatschr. f. Ohrenh. **66**:1227 (Oct.) 1932.

Morita describes a rare case of mucocele of the ethmoid cells in a girl aged 20. The symptoms, consisting of protrusion of the right eyeball, lacrimation, pain in the right eye, a heavy feeling in the head and bilateral nasal obstruction developed gradually, following a blow on the right eye five years previously. Rhinoscopic examination showed an egg-sized tumor-like formation in the right side of the nose which caused a marked deviation of the nasal septum to the other side. An

endonasal operation performed in two stages resulted in disappearance of all symptoms. The case was distinguished by the following features of particular interest: the transformation of the entire ethmoid labyrinth into a large cavity; the high grade septum deviation; the abnormal perforation of the septum by the mucocele; the simultaneous existence of the mucocele of the actual ethmoid labyrinth and of another mucocele of the "frontal ethmoid cells," the latter being separated from the former by a membrane, thus forming a two chambered ethmoid mucocele.

EDITOR'S ABSTRACT.

CLINICAL RESEARCH IN FIFTY CASES OF OZENA. J. GROFE, Ztschr. f. Laryng., Rhin., Otol. (Teil 1: Folia oto-laryng.) **23**:146 (Sept.) 1932.

Grofe summarizes the results of an investigation of fifty cases of ozena in respect to the incidence of the disease (sex, age, social class), the general condition of the patient, the type of skull formation, the anatomy of the upper respiratory passages, the degree of atrophy of the nasal conchas, the type of secretion, the presence of crusts and other findings of otorhinolaryngologic examination.

EDITOR'S ABSTRACT.

Miscellaneous

INVOLVEMENT OF EAR, ACCESSORY NASAL SINUSES AND MAXILLA IN GENERALIZED XANTHOMATOSIS (SCHÜLLER-CHRISTIAN DISEASE): PERSONAL OBSERVATION. A. GREIFENSTEIN, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **132**:337 (Sept. 21) 1932.

Greifenstein states that the knowledge of the clinical picture of generalized xanthomatosis is important to the otorhinolaryngologist because a study of the literature shows that, among twenty-six cases of the disease, the ear was involved twelve times, the maxilla thirteen times, and the nasal sinuses five times. He reports a personal observation which concerns a woman aged 42, referred to him because of disease of the left ear. The patient exhibited a total loss of labyrinthine function on the left side, and the depth of the auditory meatus was filled with granulations. Roentgenography of the skull revealed the typical osseous lesions of generalized xanthomatosis; exophthalmos and diabetes insipidus completed the triad of symptoms typical of this disease. The case was distinguished clinically by commencement of the disease with endocrine disturbance at a mature age, a protracted course of twelve years' duration and limitation of the osseous lesions to the skull. The author's conception of the labyrinthine disease as one manifestation of a progressive pathologic change of the temporal bone was supported by a roentgenologically demonstrated progressive advance of the destructive process in the temporal bone. The xanthomatous proliferation gradually extended to the auditory meatus and the labyrinth, resulting in a total loss of labyrinthine function. Roentgenography also gave evidence of proliferation into the ethmoid sinus, destruction of the alveolar process of the inferior maxilla and zones of lesser density in the inferior and superior maxilla. A severe ulcerative gingivitis existed. On the basis of a study of the literature, Greifenstein discusses the various anatomic forms of involvement of the ear in generalized xanthomatosis. In view of the recent conception of this disease as a primary disturbance of cholesterol metabolism Greifenstein calls attention to the effects of hypercholesteremia on the ear. In the case reported, roentgenization according to Sosman's method resulted in an improvement of the general condition and a tendency to regression of local changes.

EDITOR'S ABSTRACT.

Society Transactions

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

Regular Meeting, Nov. 7, 1932

CHARLES B. YOUNGER, M.D., *President, in the Chair*

OSLER'S DISEASE WITH NASAL HEMORRHAGE (MULTIPLE HEREDITARY TELANGIECTASIS). DR. FRANK J. PISZKIEWICZ (by invitation).

This condition was first put on a clinical basis by Osler in an article in 1901 (*Bull. Johns Hopkins Hosp.* **12**:330, 1901). It is a hereditary condition which is characterized by the formation of telangiectatic lesions on the mucous membranes of the mouth, nose and throat. Any mucous membrane of the body may be involved, although the disease commonly is characterized by severe epistaxis. Other hemorrhages may occur from other parts.

The diagnosis is simple, and is made by finding telangiectatic lesions in the mucous membranes, by a history of familial occurrence and by the fact that it is not limited to sex, as is hemophilia. It may be transmitted by both the male and the female of the species. Bleeding time and coagulation time are normal, although the condition has often been confused with hemophilia. The transmission differs greatly from the condition in which one sees hemophilia.

The prognosis is good if the hemorrhages occur in readily accessible areas.

The treatment consists in cauterization of the hemorrhagic points, and proper medication for the anemia that commonly is present.

This subject has been well reviewed by Goldstein, in articles which appeared in the *Medical Review of Reviews* (**37**:701 [Dec.] 1931; **38**:26 [Jan.] 1932).

DISCUSSION

DR. ALFRED LEWY: I wish to compliment Dr. Piszkiewicz on his finding and presenting this case. He showed me his patient. The disease might easily have been overlooked.

There was one phase of the disease that he did not dwell on, and that is that it may occur in the viscera and in the brain, and may be responsible on some occasions for an early demise.

DR. CHARLES B. YOUNGER: I wish to mention early in the meetings for this season that we desire to hear discussions from the younger men who attend the meetings. We are here to exchange ideas and to learn. I agree that we should encourage the presentation of unusual conditions, and we shall be glad at all times to have anything of interest presented before the Society.

I join in complimenting Dr. Piszkiewicz on his presentation.

DR. FRANK J. PISZKIEWICZ: I forgot to mention that there have been reported in the literature of the world from ninety to ninety-five families suffering from this condition. That includes about five hundred and fifty cases up to the present time.

OSTEOMYELITIS OF BONE OF FACE AND HEAD WITH RECOVERY: REPORT OF A CASE. DR. JOSEPH C. BECK.

Osteomyelitis of the face and head is not a rare condition, especially when one reviews the reports of the cases that appear in the literature, and in our society we have had quite extensive reports by Dr. Yerger, Dr. Kahn, Dr. Ballenger and

others, and tonight Dr. Galloway has presented a case. I am sure there are more cases of this disease than are reported.

I am not reporting on osteomyelitis in a general way, and not even on the acute type of osteomyelitis which often terminates fatally, but wish to confine my remarks to the cases of osteomyelitis with recovery, for that is what we are chiefly interested in — the cases in which we can make a diagnosis and save the patient. In the osteomyelitis that starts out almost like an extremely virulent type of streptococcemia, we may examine the blood and know that the patient will die, no matter what is done. Those are not the cases that I shall discuss.

Osteomyelitis about the face and head may be divided in a general way into that which follows sinus disease, whether operated on or not, and that which occurs after operations about the mastoid, the face and the head.

In reviewing the literature, it becomes evident that probably the best collection of cases is that of Tilley, and every one who writes on the subject cites his work as the best on the type of cases in which the patients recover, although he included the virulent forms of the disease. The work of Gerber stands out particularly in connection with the pathology and the pathologic entities of the disease, for there is a great difference. If one could make a biopsy and examine the decalcified specimen, one would be able to differentiate. In the cases of MacKenzie, we cannot tell whether the infection passed outward toward the periosteum or inward toward the meninges. Some of his cases had a definite picture of cessation, or limitation, and ended in fibrosis, whereas in other types the pathologic process went on diffusely and did not appear to stop anywhere.

I think that one of the finest contributions in this work is that of Fuerstenberg; he observed his cases over a long period of time. His cases have been reported clearly and definitely and are classic. One of the recent summaries you have all seen in the ARCHIVES is that by Wilensky, (**16**:160 [Aug.] 1932), but he does not go sufficiently into the subject in which we are particularly interested, that is, the intracranial complications from osteomyelitis.

I shall not at this time go into the discussion of this subject any further, for I wish to limit myself to the case I shall present, in which the patient had a most remarkable course. From this case I think we shall learn a great deal in the study of this subject. I shall only sketch the history, because Dr. Gazul will present it in detail.

In a boy, aged 7, a boil, a highly septic process, developed within the nose. The boil was opened, and following this cellulitis and phlebitis of the face, a para-orbital cellulitis and finally meningitis developed, which were treated by numerous spinal punctures before we saw him. There was a diffuse osteomyelitis on the right side of the skull, and jacksonian epilepsy developed on the left side. An emergency operation had to be performed on the skull. The arm was paralyzed and the leg paretic, and there was an intracranial complication which was suspected of being an abscess of the brain.

While the boy is in good condition at present, we are not sure that if we explore the brain we might not find an abscess. A short time after the first operation a more extensive removal of bone was necessary, although there was not so much activity at that time. Particles of bone were sent to the laboratory for microscopic pathologic study.

The question now arises as to the regeneration of bone, or the covering of the great defect by plastic repair. These are the points that are open for discussion, and now I shall show some pictures of the patient, roentgenograms and photomicrographs.

The first illustration shows roentgenograms that were taken on admission, and you can see from the front view, as well as lateral view, the various osteomyelitic areas of the deficiency of calcium. The process appears to be limited to the front and right side. The second picture shows the defect following the first operation, that is, the emergency. The third picture is a photomicrograph of the decalcified particles of bone from the most active area and shows definitely the invasion of the diploe, particularly the areas of Breschet's canal and marked leukocytic infiltration and pus. The fourth illustration or roentgenogram shows

that not sufficient bone had been removed to uncover the motor area, and also some remains of the osteomyelitic process. The fifth illustration shows the more extensive removal of the bone posteriorly and laterally, and the last illustration is that of the patient himself, showing the defect in the skull and the granulating wound. A set of roentgenograms taken today shows evidences of reformation of dense tissue over the defect. Whether there is any bone in this or not is difficult to say, it being two months and four days after the last operation.

DISCUSSION

DR. BENJAMIN GASUL (by invitation): This boy was perfectly well until June 26, 1932. Then, according to the mother's story, a swelling developed in the right nostril which extended along the right cheek and quickly involved the eye. Within two or three days the entire mouth, cheek and eye were swollen. A physician was called, who noticed that the swelling opened in the center and that a small amount of purulent discharge was present. He waited a day or two and as the boy did not improve, he sent him to a hospital on July 1. On the same day, an uncle of the patient, who was a physician, saw the boy and enlarged the original opening in the abscess.

According to the nurse's notation on the record, there was a slight bloody discharge following this. The child did not improve. There were no notations on the chart except those of the nurse, and from those we learned that from the beginning the child showed spasticity of the lower extremities and rigidity of the neck and that he had had projectile vomiting. The temperature was high. The child became delirious, and convulsions, unilateral in type, developed on July 7. The child became stuporous, and another physician was called, a former intern at Cook County Hospital, who had recently left that institution. He saw the boy on July 14 and performed a spinal puncture, which revealed cloudy fluid under greatly increased pressure, with 5,000 cells and gram-positive cocci. Spinal punctures were done at that hospital five or six times, and specimens were sent to the Roseland Community Hospital, where the pathologist found Staphylococcus haemolyticus on cultures and on smears. Another specimen was sent to the Michigan Department of Health, and they also reported a positive culture of Staphylococcus haemolyticus. A swelling was then noted in the occipital region, and the child was brought to Chicago to see Dr. Beck. He was sent to the hospital, and Dr. Beck and Dr. Guttmann asked me to assist in his care.

I have some charts showing the range in temperature and respiration during the patient's stay in the hospital which I shall show you. (Presented slides.)

The temperature ranged from 105 to 99 F. for the next three weeks.

As usually happens in cases of thrombophlebitis, there was an exudation into the medullary tissue between the inner and outer tables of the skull. If an infection extends inside, it causes pachymeningitis, and if it involves the pia, it causes leptomeningitis. According to the literature, there is much discussion regarding cases like the one presented as to whether there is a pachymeningitis first, which then extends to the inner table of the skull and then to the outer table. The cases are few, and the only way a diagnosis can be made is by necropsy. It is possible that through the hematogenous route there may be a pachymeningitis first and then an osteomyelitis. According to the nurse's report, this boy had meningitis from the time he entered the first hospital.

The prognosis is very bad. So far as we are concerned, we did not know of any patient who had recovered. We see many cases of meningitis at the Cook County Hospital, and, of course, the patients all die. Dr. Beck was very firm in his contention that the treatment should be conservative. He expressed the opinion that if the patient was operated on the day he entered the hospital he would die. He thought we should do spinal punctures, to which we all agreed, and we did a number of punctures. I shall show you a chart demonstrating the findings. (Presented slides.)

The boy went along quite well until August 13. He did show signs of increased intracranial pressure, but the temperature and pulse were decreasing. On August 13,

a convulsion, jacksonian in type, developed on the left side. He appeared to be constantly smelling something and had a projectile type of vomiting. Dr. Guttmann gave him a large dose of sodium amytal, and at that time it was decided that the child should be operated on.

An operation was performed on August 14 by Dr. Beck and Dr. Guttmann, and they removed as much osteomyelitic tissue as possible. The patient's condition became grave, and he appeared close to death while on the table. He was given fluids, gastric lavage and magnesium sulphate to relieve the intracranial pressure, and he recovered. The signs of meningitis persisted, but he got along fairly well, until he again showed marked signs of increased intracranial pressure, with severe headache and projectile vomiting. The pulse was slower. The blood pressure on one occasion was 82 systolic and 40 diastolic, and on another, 110 systolic and 84 diastolic. The patient had a definite left central facial paralysis and paralysis of the left arm and leg. What was the cause of the paralysis? The first operation was indicative of abscess, and he now had symptoms of abscess of the brain or subdural abscess. If this was the case, the thing to do was to reoperate, and we felt that a neurologist should see the patient. Dr. Lewis J. Pollock was called, and he believed that an abscess of the brain was present. Dr. Beck and Dr. Guttmann explored, but did not find an abscess. During the operation, Dr. Beck punctured the ventricle, and a large amount of spinal fluid came out, under great pressure.

Following this operation the child's condition improved. The vomiting stopped because the pressure was relieved; he did not complain so much of headache, and even the paresis was markedly improved, although he did complain of photophobia. Because he was improving and because he had been in the hospital so long, we thought it would be a good plan to send him home, with the understanding that he was not entirely well and might have to return to the hospital. He was sent home on September 15, and we continued doing the spinal punctures, for this was the only means of reducing the intracranial pressure. He got along well for a time, and again increasing signs of increased intracranial pressure developed, with an increase in the paresis and severe headache. Both Dr. Beck and Dr. L. J. Pollock were out of the city, and Dr. Guttmann and I felt that another operation was needed. We called in Dr. Hassin, who made a thorough examination and said that it was a most unusual case. We questioned Dr. Hassin as to whether the whole thing was due to pressure of the bone on the dura, but he would not commit himself, although he said it was possible. He at first advised against operation, but later agreed that it was indicated. When Dr. Beck returned and examined the child, he thought that another operation was necessary. On October 5, he operated again, and since then the boy has made a fairly good recovery. This proved the point that the pressure was sufficient to cause the hemiplegia, for following the operation the hemiplegia gradually disappeared. The vomiting stopped, and the boy has gained in weight until he weighs more than ever before, 53 pounds (24 Kg.), and has no definite symptoms of meningitis.

Concerning the osteogenesis of the bones of the skull, this case shows that osteogenesis is possible. There is now a hard mass in the area where the bone was removed. We took some roentgenograms this morning, but I think they may not show the bone formation at present, for there has not been sufficient time for calcification.

To summarize, we are presenting a case of extensive osteomyelitis of the head, probably on a hematogenous basis, with pachymeningitis and leptomeningitis in a boy, aged 7. Staphylococcus haemolyticus was found on puncture several times in the spinal fluid, in the pus from the abscess and in a smear from the dura. So far as I could tell from the pediatric literature and from our records at Cook County Hospital, this is the first patient with a case of this nature who ever has recovered.

DR. T. C. GALLOWAY: Two cases of osteomyelitis of the frontal bone, in boys 8 and 12, were seen within ten days in August, 1932, which had markedly dissimilar courses due, apparently, to the character of the invading organism.

Both cases occurred after swimming, though there had been a furuncle on the upper lid of the second boy, which apparently was not etiologically important. At operation, both patients had the findings of soft hemorrhagic bone, channels of pus in the diploe and thrombosed veins.

The first patient (R. L.) had about 30 cc. of extradural pus, but only a moderate temperature and headache. A fairly wide removal with a sharp gouge was done beyond questionable bone in a single operation, followed by irrigations with surgical solution of chlorinated soda. The patient went home on the twelfth day and rapidly recovered except for a slight discharge, which persisted from the center of the wound until a small spicule of bone was extruded two months later. The mild course was probably due to the fact that the organism was Micrococcus catarrhalis.

The second patient (J. B.) had a Staphylococcus aureus infection; he was desperately ill with high fever, and a marked mental disturbance, and for four days he was almost comatose and took food only by gavage. On the eighth day, a simple drainage operation was performed on the floor of the frontal bone, care being taken not to injure the periosteum above. A widespread removal over an area 8 cm. wide was done four days later. The dura over the left frontal lobe, which was dull, granular and covered with small collections of thick pus, did not pulsate. There was considered to be a cerebritis or a beginning abscess on account of this appearance, the marked change in temperament, a convulsion, a crossed facial and right arm spasm, the loss of all reflexes for four days and the semicoma. Grant recently emphasized the usual futility of operating before localization occurs in abscess of the brain, and expectant treatment was followed in this case. After three months this patient is apparently completely recovered except for slight drainage from the left frontal region. Latent abscess must still be considered, and further spread of infection is still possible, but the boy is active and apparently well.

The use of a bouillon filtrate of Staphylococcus aureus intradermally and as a wound dressing may have been a material factor.

A case of osteomyelitis of the maxilla in a nursling, which was also seen, is to be reported later in detail.

DR. HOWARD C. BALLENGER: In the last few years I have had three cases of osteomyelitis of the skull, all associated with acute sinus infection. Evidently one of these cases was overlooked in Dr. Gasul's search of the literature. I wish particularly to call attention to the first case, which is almost identical in every respect to the one reported by Dr. Beck. I presented the case before this Society about two years ago. It is also reported in detail in *The Journal of the American Medical Association.* I think that perhaps Dr. Gasul overlooked this case because it was reported as one of meningitis with recovery. This child had a Staphylococcus aureus infection instead of Staphylococcus haemolyticus, and it undoubtedly was a blood-borne infection, because the meningitis and osteomyelitis occurred about the same time in the early stage of the infection. The abscesses in the skull were multiple and apparently not connected. They were widely diffuse and included the frontal, the parietal and the left occipital bones. The patient recovered from the meningitis following operation on the frontal sinus, with drainage of the brain through the posterior wall of the frontal sinus. A subsequent removal of a portion of the osteomyelitic bones was performed. This was a conservative removal, the various puffy swellings being incised and scraped. The final operation on the frontal bones was done by a general surgeon and was followed by complete recovery, except for partial paralysis of some of the extra-ocular muscles of one eye.

The second case, which I have not reported before, was one of frontal sinusitis with osteomyelitis which occurred at about the same time. The patient subsequently left the city, and I received a letter from his father stating that a radical removal of the cranial bone had been done, which was followed by cerebral hernia. The last I heard, the child was alive.

The third case I think Dr. Galloway saw with me two or three years ago. It occurred in an infant with an antral infection and the rapid development of a bilateral orbital abscess. This case I reported in the *Illinois Medical Journal* as

one of orbital abscess. Acute osteomyelitis of the superior maxilla and the hard palate developed. The child died of pneumonia, and at necropsy we found diffuse osteomyelitis of the base of the skull, with an extension of the osteomyelitic process to the body of the sphenoid bone. Pus could be demonstrated around the cavernous sinus, but without involvement of the cavernous sinus itself. This case, I think, was one of infection by continuity, but the other two were probably blood-borne infections.

DR. C. F. YERGER: I have a few slides I should like to show in connection with two cases of osteomyelitis of the frontal bone, secondary to nasal sinusitis, in both of which the patients recovered six years ago. I wish to emphasize the importance of making frequent roentgenograms during the course of the disease in order to determine the presence or absence of sequestrums and the indication for sequestrectomy.

DR. HARRY L. POLLOCK: This certainly is an interesting subject, and one thing that has not yet been touched on is the real pathology. Dr. Beck stated that osteomyelitis is a somewhat rare condition, and this appears true from the many thousands of suppurative sinus cases we otolaryngologists see and in which we operate each year. When Dr. Yerger reported his original cases, I showed some slides of a patient from whom at least twenty pieces of bone had been removed from the skull. Practically the whole vault had been removed at different times within a year and a half, but the patient recovered. I saw him about a year ago and could find no evidence of dehiscence. The skull felt as though it had never been touched.

The point I wish to bring out is that most of the cases that have been reported followed operative intervention on the sinuses, chiefly the frontal, and most of them occurred in children or young adults. There is, I think, a reason for that. The diploe is much more abundant in young children, and occasionally one finds some cancellous tissues beneath the mucosa of the sinus, without any intervening compact bone. Infection of the diploe by direct continuity in these cases is easily comprehended. In one of the specimens that I have, there is a dehiscence in the posterior sinus plate of the frontal sinus through which infection would be rather simple. The consensus is that there are two methods of infection, one by continuity and one by blood stream infection. It seems to me that most of the cases are due to retrograde thrombosis (hematogenous). Those who have seen these cases know that the secondary osteomyelitic process is usually some distance from the original focus with healthy intervening bone. I think Dr. Fuerstenberg brought this out beautifully when he said that the infectious process extended extradurally, separating dura from bone with consequent loss of nutrition of the inner plate, since the main blood supply of the vault comes by way of the dura. One can take away a large portion of the external periosteum of the skull without injury to the bone, but separation of the dura from the inner table is likely to lead to necrosis. Some one stated that the outer plate is more likely to be attacked by the osteomyelitis. That is true if it progresses by continuity, but in the blood-borne infections, the inner plate has to be affected first, and the outer plate may be normal for some time during the process.

There are two types of treatment, one conservative and the other radical. The reports substantiate the fact that radical removal following localization has a much lower mortality than is present in the cases in which only conservative operations are performed. If the operation is done before the abscess localizes, the patient usually dies, but if one waits until there are definite signs of localization, the patient commonly recovers.

DR. JOHN A. CAVANAUGH: I wish to congratulate the essayist. In the case reported by Dr. Beck, I think the opening of the abscess in the vestibule was a mistake in the first place and was probably responsible for the aftermath.

There may still be a recurrence. Tilley has reported cases in which he thought the patients had recovered but in which there was a recurrence a year and a half or two years later. I believe that some types of organisms have a predilection for certain structures and that that accounts for the development of the osteomyelitis.

The remarks of Dr. Pollock regarding conservative and radical treatment should be borne in mind. I am much in favor of the conservative type of treatment, waiting for localization and taking steps cautiously.

DR. JOSEPH C. BECK: I thank the gentlemen for their liberal discussion and for bringing out the important points, which I did not take time to do.

Dr. Gasul and Dr. Galloway mentioned the infants who have these lesions. In my experience I have seen perhaps ten or more cases of this type in new-born infants or in children in early infancy, with an infection apparently in the nose. In these cases there remains a nidus of infection somewhere, and there may be repeated flare-ups. This was true in the second case I illustrated.

We have often discussed the question of squeezing acute boils or furuncles of the nose, and this may have been the cause of the trouble in the second case. The child's temperature was high, and it was felt that the lesion should be opened. In the literature, a great deal of credit is given the hot lamp in frontal sinus disease, as well as in other conditions. While it may not have any specific effect, while waiting for the lesions to develop, this treatment is used.

Dr. Cavanaugh brought out the question of the type of bacteria present, and I think that this is an important point, as was brought out in the discussion. In some cases the patients are doomed from the start because of the virulence of the infection.

Dr. Pollock mentioned a case in which there was involvement of practically the entire skull, with sections of normal bone between. Those cases are due to infection by continuity, rather than by retrograde thromboses.

There is no one type of treatment in these cases, and I am sure every one will be guided by the findings in each individual case.

The point brought up by Dr. Pollock as to why we do not see more of these cases of osteomyelitis is a good one. I think that in many cases it must be a question of the circulation. In some persons the osteomyelitis does not progress, and in other cases it goes on no matter what is done.

SUBMUCOUS RESECTION OF THE SEPTUM ON THE CADAVER AND THE LIVING. R. H. GOOD.

One feels inclined to apologize for writing on this subject, as the literature for the past thirty years is full of excellent descriptions of this operation. However, I have hospital records of more than two thousand cases, and I am convinced more than ever that there is no end to the new problems that one encounters. The subject is a life study.

My son, Dr. Palmer Good, and I have made a moving picture of this operation on the cadaver and on the living subject for teaching purposes and are presenting it before this society. In the operation on the living, no one but the operator can see what is being done, and if he still uses the ordinary head-mirror he must do some of the work by the sense of touch. Thus, the student has no opportunity of actually seeing what is being done.

As I travel about the country from time to time, I take the opportunity of going to hospitals for diseases of the eyes, ears, nose and throat to see operations. One is amazed at the number of rhinologists who are still inadequately informed on this subject. There are many who do not know how to anesthetize the septum so that it is painless and practically bloodless. Many times there is a lack of proper instruments to correct complicated deformities of the septum. The majority of rhinologists still use the head-mirror, which is not designed for looking into deep, narrow cavities.

The head-mirror cannot be used for bronchoscopy because the rays of light are not parallel. Neither can one see through a one-fourth inch (0.64 cm.) tube 5 inches (12.7 cm.) long. The same holds true in an operation on the septum, as the space is narrow and long.

There are still many operations on the septum requiring from one to three hours, because the operator cannot see what he is doing far back in the nose. An

operating light with parallel rays enables one to see into small, remote cavities, enabling the surgeon to see exactly what he is doing.

Two weeks ago a patient who had had three operations on his septum and still had trouble in breathing consulted me. He had interviewed a number of rhinologists, and all had advised another operation. On examination, I found the remaining cartilage up above deflected to the right and resting against the outer wall of the nose. There was a posterior spur, which pressed into the posterior third of the inferior turbinate. On the left side, there was a large spur near the floor of the nose, adherent to the inferior turbinate. I assured the patient that the only thing I could do would be to reoperate on his nose. He replied that he could not endure another operation as the others had been so painful. After assuring him that he would have no pain, he agreed to have it done. He doubted my word, and during the preparation he was in a most nervous condition. After I started operating he was relieved of his anxiety and fell into a jolly conversation during the remainder of the operation, and he assured me that he had no pain.

I have seen a number of operators who stopped in the midst of an operation and told the patient to return later to finish the operation because of the severe pain and excessive bleeding. Such bleeding frequently occurs in cases in which the nose has not been completely anesthetized.

Hardly a week goes by but that some patient will say that her physician told her that operations on the septum are not satisfactory. I know of no operation that gives more relief when properly done.

I, therefore, do not feel an apology necessary if I can help some one with the aid of the moving picture and this article to do the operation better and in much less time.

Exhibit A on the cadaver was done in the department of otolaryngology of Rush Medical College through my son, Palmer. The photographic illumination is 1,500 cc. distributed at two angles, and the parallel-ray headlight throws 1,000 lumens of light directly on the field of operation. With the aid of such lighting, we are able to take movies of cavities such as the throat for tonsillectomy.

I shall give a short résumé of the operation.

The patient receives a hypodermic of ¼ grain (0.016 Gm.) of morphine and 1/150 grain (0.0019 Gm.) of atropine one-half hour before the operation. The nose is sprayed sparingly with 5 per cent cocaine in fresh epinephrine, 1:1,000. Flaky cocaine in epinephrine chloride, 1:1,000, is applied twice to the entire septum and the area of the sphenopalatine ganglion. These applications are given about two minutes apart. The face and the anterior nares are sterilized with 50 per cent alcohol.

I wish to make it emphatic that it is most essential to have fresh epinephrine chloride if bleeding is to be controlled.

The patient lies flat on his back on the operating table, with a pillow under his head. This position eliminates almost entirely all toxic symptoms, and the patient is infinitely more comfortable. This is all made possible by the use of an operating headlight with parallel rays.

Furthermore, the surgeon can work with much greater ease when having his hands down, there being more blood in the hands and, therefore, a better sense of touch. This is especially true as we get older. The incision is made on the side most liable to perforate. The knife is held obliquely to the septum, and the incision is made through the membrane and perichondrium and partly into the cartilage, but not through the cartilage. The incision begins high up and extends down to the floor of the vestibule. The distance from the tip of the nose depends on the nature of the deflection.

The Freer elevator is used to begin the dissection of the membrane back to the ethmoid plate, and this is done only in the upper half of the incision. We now use the Pierce elevator and dissect the membrane back and up and forward. Then the Pierce elevator is reversed, and the lower half of the entire septum is dissected from behind, forward.

The incision through the cartilage is usually made with the little finger held in the opposite nostril. The same procedure as that just described is used in dissect-

ing the opposite membrane. Rogers' elevator is used to dissect the membrane from spurs and deeply curved recesses.

My retaining septal speculum is used to separate the membranes, which eliminates the need of assistants.

Ballenger's swivel knife removes the cartilage. Good's nasal chisel, which has blunt corners, is used to chisel the crest and ethmoidal plate above.

Bone forceps are used to break and remove necessary bony parts. The operator should be sure not to leave projecting cartilage or bone that is not in the midline. Tincture of iodine is applied along the line of incision, and one side is packed with a finger of cotton in which a narrow strip of gauze is inserted. This keeps the membranes together and is not for the purpose of preventing hemorrhage.

DISCUSSION

DR. G. J. MUSGRAVE: I wish to congratulate Dr. Good on his presentation and the Society on hearing him. I think that this is a stimulating thing for teaching students. I think I saw my first submucous resection with Dr. Good. He did it thoroughly, but I was unable to get the principles of submucous resection that many of the young men in the room can get now, with perhaps one demonstration.

There is not much to say in regard to the principles involved which have not been presented. There are many modifications of this procedure, and each of us has his own pet method. I was impressed with Dr. Good's emphasis of the fact that in order to do any nasal operation it is important to secure the patient's complete confidence and cooperation.

I have been satisfied with my methods of anesthetizing these patients. I noticed that Dr. Good spoke of having his patients go to the hospital some time before the operation. I insist that my patients go to the hospital the night before I intend to operate, and I give them sodium amytal on retiring. In the morning they are given, hypodermatically, ¼ grain (0.016 Gm.) of morphine and 1/100 grain (0.0006 Gm.) of scopolamine. I formerly gave three doses of ⅙ grain (0.01 Gm.) of morphine and 1/150 grain (0.0019 Gm.) of scopolamine, but got depressing results. I have found that one dose is sufficient to take away the dread of the operating room and to obtain the synergistic effect with cocaine.

There is one point in the way of technic that I have departed from during the last two or three years, and I should like to hear some comment on it. We all know the danger that is associated with interfering with a furuncle in the vestibule of the nose. At our last meeting we had a stimulating discussion regarding the bacteria of the nose. At that time I was impressed by the fact that possibly this thing that I had been thinking was so good is not without danger; namely, in the last few years I have been making my initial incision in the skin of the vestibule just above the columella. This insures a flap that does not tear. In view of these observations, is this procedure accompanied by the same danger that occurs with surgical intervention of an infection at this point?

DR. JOHN A. CAVANAUGH: I wish to congratulate Dr. Good on his work, and I believe that, with a few changes, his method of presentation by means of the movie will be a valuable aid in teaching.

I should like to emphasize conservatism. In a case I saw recently, the patient complained of a flapping sensation in the nose, so disagreeable that it was driving him distracted. It required perforating the septum. Perhaps some of you will remember a paper I read before this society on submucous resection. The operation described was something like this (illustrating on blackboard). I use my special instruments for taking care of these various deformities. The usual incision is made, elevating the mucous membrane only on the convex side and removing small strips of cartilage with the septum shave. If a ridge or elevation is present, it is handled in the same manner, and if the deflection occurs over the bony premaxillary wing, the initial incision is made through the cartilage. It is then possible to move the cartilage into the median line and still maintain the normal

or practically normal nasal septum. If there are scarred areas on the septum, one can go around them.

Dr. Good's technic might be efficacious if the object is to remove all the bone and cartilage of the septum, but I plead for conservatism.

DR. FRANCIS LEDERER: I am sure we are agreed that Dr. Good's technical skill in removing the septum is all that it should be, but I think he is looking for constructive criticism as to the teaching value of his presentation. I think that as the picture now stands it has no teaching value whatsoever. You who are here can anticipate every little point and appreciate each step that is made in the operation, but, if I recall correctly, some years ago Dr. Beck made a motion picture of the actual operation on the mastoid. As a teaching aid it had no value whatever, and he came to the conclusion that diagrammatic illustration is much better, and in consequence he made such a film, which was more practical. I do not believe that any student could be taught from this motion picture. I think that interspersed with each operative step on the picture there should be a diagram illustrating the anatomy and the technic. It should say what is done with each instrument, rather than stating the name of the instrument. I think that it is much more valuable to have diagrammatic illustrations, and that they should be interspersed with the operative procedure as illustrated on the cadaver. I have no doubt that the technic shown is excellent so far as photography is concerned. It merits our praise, and if the authors do the things that I have suggested, the picture should be of greater teaching value.

Furthermore, I think that we should get away from the use of men's names in connection with instruments. I know Dr. Good likes to give priority where it belongs, but I think that he should tell what is done with the instrument rather than the name of the inventor.

DR. AUSTIN A. HAYDEN: I was much interested in Dr. Good's motion picture and remarks, and particularly in the experience of the professor in the midwest medical college who advised the patient to come back for completion of his submucous operation another day. In my own acquaintance there is at least one man of national reputation who has never yet done a submucous resection. Turbinate resection is the only means he employs for the relief of symptoms for which we commonly do a submucous resection.

While I appreciate the work the Drs. Good have done in devising their headlight, I do not believe that it is superior to the ordinary head-mirror or that with the latter the interior of the nose cannot be adequately explored.

Concerning the one hour operations, I wish to tell you that the first submucous operation I saw lasted not one hour but three, and the patient was reanesthetized three times during the course of the procedure. I have seen that septum within the last week. It was done on a person from my home town in Wisconsin, by Dr. Otto Freer himself. I speak only with the greatest admiration for Dr. Freer. He did the work painstakingly and excellently under the great handicap of a high myopia.

I have made some motion pictures myself and appreciate thoroughly the great amount of time that has been spent on this picture. May I offer some friendly suggestions? First, the titles Dr. Good uses can be greatly improved in their construction. The best titles I have been able to secure have been from the Eastman Company. I have tried making them myself and found this unsatisfactory. Eastman titles are uniform, inexpensive, plainly printed and so arranged that the maximum number of letter spaces can be easily seen. It must be remembered that titles shorten the footage that can be used for exhibition of the real subject matter that is being presented. As Dr. Lederer suggests, talkies will help in this respect.

The second general criticism of Dr. Good's photography that I would give is that the field is too large and the detail correspondingly lacking. The first part of the picture (supersensitive film) was overexposed, and it must be remembered that in photography dark fields must be overexposed to bring out details. In the last part (Standard Panchromatic film), the detail did not show well. This I think was properly exposed, but should have been overexposed so that the mucous

membrane would appear gray rather than black. In regard to photographing the instrument table and the patient, they were working in each instance against a white background on which the instruments did not sharply contrast, so that the details of the instruments are much lessened by the fact that the background was white. A much better picture could have been made if a gray sheet had been used on the table instead of a white one.

In regard to trying to show a photograph of the technic of the submucous resection on a living patient, you can readily see that it is impossible, no matter what light is used. A general picture of the patient and the operation only can be taken. Dr. Good did very well, therefore, to portray first the steps of the operation on the open skull.

The last thing I wish to mention is in regard to what Dr. Cavanaugh said. I noticed the specimens Dr. Good showed on the screen and passed around, and the very thing Dr. Cavanaugh mentioned occurred to me in regard to the resection. A patient who has had that sort of resection often will be less comfortable following the operation than he was before, owing to the flapping of the resected flabby septum. Dr. Cavanaugh's idea of leaving a framework of bone is good. There are cases, however, in which it is difficult to do this, and if an attempt is made the original obstruction will have to be left intact. For that reason the procedure I have used with great satisfaction is to remove the septum completely, as Dr. Good has done, and then take the cartilage and bone, principally cartilage, and shape it on the instrument table on a wooden board, trimming it up so that it will be straight, and then reinsert this into the septum. In that way you have a septum that will not flap and a satisfactory nose.

In regard to the anesthesia, I know that this is an important detail, and I have no doubt that the patient Dr. Good operated on was grateful for being relieved of the pain he complained of in the previous three operations. I do not believe, however, that it is well to put cocaine in the nose under any consideration with a spray, as Dr. Good suggested. The possibility of a certain amount of the cocaine going back into the throat and causing cocaine poisoning is always to be considered. The flake method is widely used, and I have used it myself, but I think packing the nose with pledgets of cotton wrung dry from a solution of epinephrine and cocaine and left in for ten minutes or so will provide anesthesia that is complete, and will avoid the possibility of the patient swallowing any cocaine.

DR. JOSEPH C. BECK: I rise particularly to challenge the statement of Dr. Hayden in regard to replacing material removed from the septum, and not telling us what he finds from three to six months later. It has been shown that this is not good practice; the replaced particles separate the perichondrium on both sides and do not grow again, and produce, perhaps, a worse deformity than the original one. From my experience, I would advise not replacing bone or cartilage.

I wish to compliment Dr. Good for spending time and money on photography of this type. I have had my fill of that and still find it unsatisfactory for good teaching purposes. We have to add illustrations and graphic diagrams to the pictures, as Dr. Lederer brought out.

In connection with mopping the nose with the patient in the recumbent position, as was shown in the picture, I wish to know why suction was not used instead. I am sure that the pictures would be better under those circumstances.

DR. G. HENRY MUNDT: I think that the first picture of a nasal surgical procedure I ever saw was presented about fifteen years ago by Dr. Good, and I believe that it is partly responsible for Dr. Good's placing the names of the instrument on the film.

I wish to compliment Dr. Good, Jr., on the development of the excellent light apparatus. I can understand why Dr. Robert Good says a head-mirror is of no value, for after one has accustomed oneself to a light such as his son has devised one realizes how valuable it is. There is no question regarding its superiority for nasal surgical procedures, for mastoid surgical procedures or for any other surgical intervention we do. There is no question about its illuminating value.

I am glad that Dr. Hayden brought out the point about spraying cocaine into the nose. That I have never been able to see. I was surprised that Dr. Good insisted that the patient should be thoroughly anesthetized, but did not tell us the procedure. I send patients to the hospital in the morning and operate in the afternoon, using 3 grains (0.19 Gm.) of pentobarbital sodium thirty minutes before operation; this is the short-acting barbital preparation. I used for a long time cocaine crystals, but am away from that method now.

I think that the picture is valuable. The first one Dr. Good showed was probably fifteen years ago, and he was criticized because he could not show the details.

I wish to assure Dr. Cavanaugh on one point, and that is that many submucous resections are done in which it is not necessary to do a subsequent perforation of the septum.

DR. OTIS H. MACLAY: I have always been much interested in this subject. We have not had many papers in this connection for some time, and if this presentation does nothing more than enliven the subject, it is worth while. I do not believe that the young men have been getting the amount of experience and instruction during the last ten years that they should have in nasal surgical procedures. The ear and its problems have become so prominent that perhaps the nose has been somewhat neglected.

Dr. Freer emphasized the point that we should never remove any more septum than was absolutely necessary, and we should not leave a septum with a large flap that will make trouble later.

The sedative preparation I have been using before operating is the pentobarbital of which Dr. Mundt spoke. It does away with the toxic effects of cocaine, and eliminates the nervousness these patients often have. The time spent in operating is, I think, an entirely nonessential affair, except that we should do the work thoroughly and carefully.

Complications should be mentioned. Postoperative hemorrhage, an infection or both may occur in the same case. Under such conditions, the flap must be opened and appropriate measures instituted as soon as possible.

DR. ROBERT GOOD: Dr. Musgrave has brought out an important point about the use of scopolamine in operations on the nose, especially the repeated dose. I have had several cases in which I was so alarmed that I have stopped the use of scopolamine in operations on the nose, but not in other operations.

I recall one patient whose nose I packed on both sides, and to my surprise when I went to her room before leaving the hospital she had stopped breathing entirely. I quickly opened her mouth, and after a few minutes she started to breathe. It is my opinion that this patient was so completely under the influence of scopolamine that she did not have enough energy to open her mouth and breathe.

I wish to emphasize the need of a nurse remaining at the bedside of the patient until all danger is past if scopolamine, sodium amytal or sodium barbital is used.

Dr. Cavanaugh has interpreted this moving picture as conveying the idea that we remove all the septum, which is far from the fact, but we do insist that every projecting part not in the midline be removed.

About seven years ago Dr. Cavanaugh gave an excellent paper stressing conservatism in operations on the septum. One cannot but admire such efforts. However, as long as fifteen years ago I went through the experience of being conservative in a number of cases. To my surprise, on seeing these patients a month or more after the operations, I found projections staring me in the face, which defeated the purpose of the operation.

Dr. Beck has spoken well about cartilage implants which are absorbed and leave a thickened membrane. However, if the perichondrium is left attached on one side of the cartilage, it will not absorb. A complete resection of the septum does not cause flapping if it is in the midline, allowing the same amount of air to pass on each side, but it will flap if a deviation is not corrected in the posterior part of the nose which keeps the membrane more to one side. This omission is

caused by the ordinary head-mirror, which is inadequate to illuminate the deep recesses of the nose.

Dr. Cavanaugh mentions dissecting around scarred or ulcerated areas of the nose, leaving the cartilage intact at that point. It is this so-called ulcer or scar which is the common place for nose bleeding. I have performed a number of resections of the cartilage in this area just to stop nose bleeding. It is the best treatment I know for severe hemorrhages from this ulcer. I would add that one of the main purposes of a submucous resection is to remove the cartilage opposite this finger trauma area due to stoppage of the nose.

Dr. Lederer thinks the movie inadequate for teaching without diagrams and models, and I heartily agree. On the subject of naming instruments, I would say that when I brought out my rasp, in 1906, for frontal sinus and antrum operations, it was only about three months later that a specialist in Detroit made a copy of this rasp and called it his own. In a year or two there were more in Chicago who made rasps with the identical handle and called it theirs. Hours and often years of work are spent on inventing an instrument, and it deserves the inventor's name.

I have covered Dr. Hayden's remarks on the head-mirror in my paper. His remarks on the picture are well taken, and my son is well aware of the imperfections, which will be remedied as experience is accumulated.

I am opposed to the method of applying cotton pledgets of cocaine to the septum for anesthesia. This leaves a limitless supply of cocaine to be absorbed into the system for ten or fifteen minutes, when all we want is to anesthetize the membrane. These pledgets of cotton are sometimes lost in the nose, especially when one hopes to find them with a head-mirror.

In the London Eye, Ear, Nose and Throat Hospital, in a room adjacent to the operating room, I saw a lad lying on a table, and he was as white as a sheet. I asked what was wrong, and the nurse replied that his nose was packed with cotton pledgets of aqueous cocaine for an operation on the septum. The physician was delayed, and the pack was left in the nose for nearly an hour. A procedure such as this is dangerous. It would even be dangerous if the cocaine were used with epinephrine.

Dr. Mundt evidently has purchased many lights and discarded them all for a parallel-ray head light. In time head-mirrors will be a thing of the past.

Obituary

GEORGE FETTEROLF, M.D., Sc.D.

1869-1932

George Fetterolf died at his home in Philadelphia on Dec. 29, 1932. He is survived by his wife, Lila Prosser Fetterolf, and a brother, Edwin, the well known architect. He was born at Collegeville, Montgomery County, Pa., on March 31, 1869. His father, Prof. Adam H. Fetterolf, A. M., Ph.D., was at that time located at Freeland, now Collegeville, Pa., as head master of Freeland Seminary, later incorporated as Ursinus College. When George was about 1 year old, Professor Fetterolf with his wife, Annie H. Fetterolf, moved to Andalusia, Pa., establishing there a private school. This he discontinued later to accept the presidency of Girard College, serving in this position continuously for twenty-seven years.

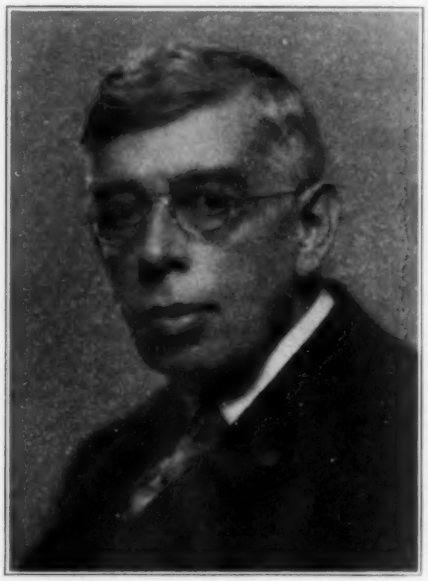
The effect of the atmosphere of literature and learning of education and culture that surrounded George Fetterolf as a child, is obvious throughout his life. The education begun in his father's school was continued in the Episcopal Academy of Philadelphia, preparatory to entering the University of Pennsylvania, in 1883. With this institution he was associated in various capacities until his death, almost half a century later. Graduating with the degree of A.B. in 1887, he became demonstrator of histology and embryology, in the Biological School. He received his degree of M.D. from the Medical School in 1891. For twenty-three years he was associated with the department of anatomy, serving as prosector, demonstrator and instructor, and for nine years as assistant professor. In 1924, he was elected professor of otolaryngology, a chair he occupied until his death.

In addition to his services at the University of Pennsylvania Hospital, he was on the staff or consulting staff of many other institutions, notably the Philadelphia Polyclinic, the Henry Phipps Institute, the Whitehaven and Eagleville sanatoriums, the Eastern Pennsylvania Institute for the Feeble-Minded and and Epileptic, the Methodist Episcopal, Kensington and Shriner's hospitals.

He served with honor as a major in the Medical Corps of the United States Army, 1918-1919, being stationed at Fort Hancock, Augusta, Ga.

His interest in his fellow man naturally resulted in many social activities. He was a much appreciated member of many clubs and fraternal organizations.

He was an outstanding member of the leading medical organizations, notably, the American Medical Association, the American College of Surgeons, the American Association of Anatomists, the American Laryngological, Rhinological and Otological Society, the Philadelphia College of Physicians, the Philadelphia Pathological Society. For ten years he was treasurer of the American Laryngological Association, serving not merely as a custodian of the funds but as a wise and efficient member of the council. He resigned the treasurership to accept the presidency of the association; this office he held at the time of his death.



GEORGE FETTEROLF, M.D., Sc.D.
1869-1932

In 1929, he was elected by the Board of Trustees of the American Medical Association as a member of the Editorial Board of the ARCHIVES OF OTOLARYNGOLOGY; in this position also he was rendering faithful service up to the onset of his final illness.

Dr. Fetterolf was a keen observer and a great clinician, and he was born and grew up and lived in a literary atmosphere. As a natural sequence, it is especially for his scientific research and literary work that he will have a permanent place in medical history.

Most of his many and invaluable literary contributions to medical science were based on his profound knowledge of human and comparative anatomy, and in later years were concerned chiefly with the problems of his chosen specialty, otolaryngology.

The following list of his scientific writings is by no means complete, but will serve to show the character of his life's work:

The Rudimentary Pineal Eye of Chelonians, Naturalist **21**:1126, 1887.

A Simple Method of Correcting Certain Deformities of the Nasal Septum, Laryngoscope **12**:584 (Aug.) 1902.

Reflex Cardiac Inhibition Resulting from Irritation of the Peripheral Fibers of the Tri-Facial Nerve and Occurring in the Course of an Operation for Chronic Empyema of the Frontal Sinus, Tr. Am. Laryng., Rhin. & Otol. Soc. **10**:409, 1904; Ann. Otol., Rhin. & Laryng. **13**:58, 1904.

Variations in the Arteries of the Human Body, Univ. Pennsylvania M. Bull. **21**: 323 (Jan.) 1908-1909.

The Anatomic Explanation of the Greater Amount of Vocal Fremitus and Vocal Resonance Normally Found at the Apex of the Right Lung, Arch. Int. Med. **3**:23 (Feb.) 1909.

Variations in the Muscles and Nerves of the Human Body, Univ. Pennsylvania M. Bull **22**:174 (July) 1909.

Compression of the Pulmonary Veins, the Pressure Factor in the Etilogy of Cardiac Hydrothorax (in collaboration with H. R. M. Landis), Am. J. M. Sc. **138**:712 (Nov.) 1909.

Some Anatomic Features of the Child's Thorax and Their Practical Application in Physical Diagnosis (in collaboration with J. Claxton Gittings), Am. J. Dis. Child. **1**:6 (Jan.) 1911.

The Anatomical Explanation of the Paralysis of the Left Recurrent Laryngeal Nerve Found in Certain Cases of Mitral Stenosis (in collaboration with George William Norris), Am. J. M. Sc. **141**:625 (May) 1911.

The Relief of Pain in Advanced Tuberculosis of the Larynx by Means of Injections of Alcohol into the Internal Laryngeal Nerve, Ann. Otol., Rhin. & Laryng. **21**:128 (March) 1912.

The Value of Frozen Sections in the Teaching of Physical Diagnosis (in collaboration with H. R. M. Landis), Am. J. M. Sc. **143**:495 (April) 1912.

The Anatomical Explanation of the Relatively Less Resonant, Higher Pitched Vesiculotympanitic Percussion Note Formally Found at the Right Pulmonary Apex (in collaboration with George William Norris), Am. J. M. Sc. **143**:637 (May) 1912.

The Anatomy and Relations of the Tonsil in the Hardened Body, with Special Reference to the Proper Conception of the Plica Triangularis; The Principles and Practice of Tonsil Enucleation as Based Thereon, Am. J. M. Sc. **144**:37 (July) 1912.

The Topography of the Cardiac Valves as Revealed by the X-Rays (in collaboration with George William Norris), Am. J. M. Sc. **145**:225 (Feb.) 1913.

The Larynx in One Hundred Cases Dying of Pulmonary Tuberculosis: A Clinical Postmortem Study, Laryngoscope **26**:37 (Jan.) 1916.

A Case of Vagotonia, Apparently Originating in the Nasal Accessory Sinuses, Ann. Otol., Rhin. & Laryng. **25**:587 (Sept.) 1916.

A Study of the Topography of the Pulmonary Fissures and Lobes in Infants (in collaboration with J. Claxton Gittings and A. Graeme Mitchell), Am. J. Dis. Child. **12**:579 (Dec.) 1916.

Serial Frozen Sections of the Thorax from a Case of Aneurism of the Aortic Arch (in collaboration with George William Norris), Ann. Otol., Rhin. & Laryng. **26**:710 (Sept.) 1917.

The Otolaryngologic Features of the Influenza Epidemic at Camp Hancock, Ga., September-December, 1918 (in collaboration with William J. Rideout), Ann. Otol., Rhin. & Laryng. **29**:44 (March) 1920.

A Case of Post-Tonsillectomy Pulmonary Abscess, Laryngoscope **32**:517 (July) 1922.

The Safeguarding of the Tonsil and Adenoid Operation, Am. J. M. Sc. **164**:884 (Dec.) 1922.

The Reaction of the Paratonsillar Tissues to Tonsillectomy (in collaboration with Herbert Fox), Am. J. M. Sc. **166**:802 (Dec.) 1923.

The Use of the Galvano-Cautery in the Treatment of Tuberculosis of the Larynx, Laryngoscope **34**:290 (April) 1924.

Ephedrin Sulphate, the Alkaloid of MaHuang (in collaboration with Marshall B. Sponsler), Arch. Otolaryng. **2**:132 (Aug.) 1925.

Disease of the Upper Respiratory Tract in Relation to the Etiology and Treatment of Bronchial Asthma (in collaboration with Simon S. Leopold), Atlantic M. J. **30**:286 (Feb.) 1927.

A Comparison of the Outlines of the Frontal Sinus in Vivo as Shown by Transillumination and the Roentgen Rays, Arch. Otolaryng. **9**:181 (Feb.) 1929.

A Case of Infection by Brucella Melitensis, Var. Abortus, Complicating Tonsillectomy, Ann. Otol., Rhin. & Laryng. **38**:675 (Sept.) 1929.

Book Reviews

Blind Flight in Theory and Practice. By William C. Ocker, Major, Air Corps, United States Army, and Carl J. Crane, First Lieutenant, Air Corps, United States Army. Price, $3. Pp. 200. San Antonio, Texas: Naylor Printing Company, 1932.

Major Ocker has been a foremost pioneer of the art of flying blind in fog or in a hooded plane, relying entirely on instruments. This interesting and readable book thoroughly covers the subject, most of its two hundred pages being devoted to a discussion of the various instruments available and their use in blind flight. The introductory chapter contains a brief historical review, and the second chapter discusses briefly the function of the various senses in orientation in space with particular reference to the vestibular apparatus.

In the early days of aviation it was assumed that a normally functioning labyrinth was necessary for successful flying. The authors point out that, on the contrary, a flier must, first of all, learn to disregard the false impressions given by the internal ears during flight. For example, in executing a banked turn the centrifugal force plus gravity gives, to the beginner, the sensation that the earth is tilted at an angle. The flier soon learns to compensate for this false sensation by his visual impressions. In blind flight these visual impressions are absent, and the aviator must learn to rely on his instruments to the exclusion of his false labyrinthine stimulations.

The interesting observation was made by Lieutenant Crane that blindfolded pigeons released in midair lose all flight control and perform spiral dives, stalls and various other evolutions until, finally holding their wings at a high dihedral angle, they descend to the ground much like a parachute. The most important contribution to blind flight has been the invention of the artificial horizon which provides the flier with the visual impression to which he is accustomed. The importance of blind flight lies chiefly in the ability to navigate in fogs, clouds and storms, which heretofore have accounted for a third of all accidents in commercial aviation. The time will soon be at hand, according to the authors, when all commercial planes will be equipped and all commercial pilots trained for blind flight.

L'oreille et des maladies; l'audition et ses troubles; les organes de l'équilibre. By A. Bonain. Price, 45 francs. Pp. 294, with 153 figures, 20 in color. Paris: Gaston Doin, 1933.

The author has undertaken to present in a rather novel manner the diseases of the ear. The novelty consists of a much more exhaustive presentation of the anatomy and physiology of the acoustic portion as well as of the mechanism of equilibrium. He has utilized as his illustrations, for the most part, selected drawings from the outstanding works on anatomy, gross as well as microscopic. He shows an appreciation in his selection of illustrations of those which clarify rather than confuse the reader. The gross anatomy of the temporal bone, as well as the histology of the end-organs in the labyrinth, is so complicated that the details need clarifying by a more or less schematic presentation, and this the author has given admirably. He devotes 158 pages of a total 294 to this presentation of the fundamentals of otology. It would seem apparent to any one who has looked over the textbooks in English on diseases of the ear that most of these lack pitifully an adequate presentation of the fundamentals and that too much has been devoted to the purely clinical. The volume contributed by Bonain makes a valuable addition which every one interested in the organ of hearing should read.

A Standard Classified Nomenclature of Disease. Compiled by The National Conference on Nomenclature of Disease. Edited by H. B. Logie, M.D. Price, $3.50. Pp. 700. New York: Commonwealth Fund, 1933.

This is a volume of seven hundred pages, but it is printed on extra thin paper and of a size that can be slipped into one's coat pocket. It has been compiled with the active cooperation of twenty-seven national societies and governmental bureaus in clinical and statistical fields. It presents a numerical index of diseases and takes into systematic account both the etiology of the disease and the part of the body affected. It is the first classified nomenclature developed by national rather than local interests. It is hoped that this standard nomenclature will promote uniformity in the expression of medical thought and encourage greater precision in the analysis of causes of illness and death by providing a more accurate basis than any heretofore available for mortality and morbidity statistics and for recording clinical experience. The price of the book is approximately the cost of printing and distribution without any regard for the expense of compilation.

Directory of Otolaryngologic Societies *

INTERNATIONAL

Second International Otolaryngological Congress

President: Prof. Dr. A. G. Tapia, Hermosilla, 2, Mod, Madrid, Spain.
Secretary: Prof. Dr. C. E. Benjamins, Verlengde, Heereweg 143, Gronigen, Holland.

FOREIGN

New Zealand British Medical Association, Ear, Nose and Throat Section

Chairman: Dr. James Hardie Neil, Auckland.
Secretary: Dr. W. Gillies Borrie, Auckland.

Royal Society of Medicine, Section of Laryngology

President: Dr. E. Musgrave Woodman, 22, Newhall St., Birmingham, England.
Secretary: Dr. A. Lowndes Yates, 33 Weymouth St., W. 1, London.
Place: London. Time: May 5, 1933.

Royal Society of Medicine, Section of Otology

President: Dr. J. F. O'Malley, 6, Upper Wimpole St., W. 1, London.
Secretary: Dr. C. Gill Carey, 106, Harley St., W. 1, London.

NATIONAL

American Medical Association, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Harris P. Mosher, 828 Beacon St., Boston.
Secretary: Dr. John J. Shea, 1018 Madison Ave., Memphis, Tenn.
Place: Milwaukee. Time: June 12-16, 1933.

American Academy of Ophthalmology and Oto-Laryngology

President: Dr. B. R. Shurley, 62 W. Adams Ave., Detroit.
President-Elect: Dr. J. M. Wheeler, 30 W. 59th St., New York.
Executive Secretary: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.
Place: Boston. Time: Sept. 18-22, 1933.

American Bronchoscopic Society

President: Dr. Richmond McKinney, 899 Madison Ave., Memphis, Tenn.
Secretary: Dr. Edwin McGinnis, 104 S. Michigan Ave., Chicago.

American Laryngological Association

President: Dr. George Fetterolf, 2010 Spruce St., Philadelphia.
Secretary: Dr. George M. Coates, 1721 Pine St., Philadelphia.
Place: Washington, D. C. Time: May 8-10, 1933.

* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY

President: Dr. Joseph C. Beck, 185 N. Wabash Ave., Chicago.
Secretary: Dr. R. L. Loughran, 33 E. Sixty-Third St., New York.
Place: Chicago. Time: June 8-10, 1933.

AMERICAN OTOLOGICAL SOCIETY

President: Dr. George L. Tobey, Jr., 270 Commonwealth Ave., Boston.
Secretary: Dr. Thomas J. Harris, 104 E. Fortieth St., New York.
Place: Washington, D. C. Time: May 2-3, 1933.

SECTIONAL

EASTERN NEW YORK EYE, EAR, NOSE AND THROAT ASSOCIATION

President: Dr. R. H. Seeley, 9 Court St., Rutland, Vt.
Secretary-Treasurer: Dr. Arthur F. Holding, 142 Washington Ave., Albany.
Time: Third Wednesday of October, November, March, April, May and June

NEW ENGLAND OTOLOGICAL AND LARYNGOLOGICAL SOCIETY

President: Dr. Henry A. Barnes, 520 Commonwealth Ave., Boston.
Secretary-Treasurer: Dr. Lyman G. Richards, 319 Longwood Ave., Boston.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY

President: Dr. Hans Barkan, 490 Post St., San Francisco.
Secretary-Treasurer: Dr. Frederick C. Cordes, 384 Post St., San Francisco.
Place: San Francisco. Time: June 28-30, 1933.

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. H. R. Secoy, 1802 Hewitt Ave., Everett, Wash.
Secretary-Treasurer: Dr. Francis A. Brugman, 806 Cobb Bldg., Seattle.
Time: 8 p. m., third Tuesday of each month, except July and August.

SAGINAW VALLEY ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. R. B. Smith, Alma, Mich.
Secretary-Treasurer: Dr. W. K. Slack, 308 Eddy Bldg., Saginaw, Mich.
Place: Saginaw, Mich., or Bay City, Mich. Time: First Friday of each month except July and August.

SIOUX VALLEY EYE AND EAR ACADEMY

President: Dr. John B. Gregg, 101 S. Main Ave., Sioux Falls, S. D.
Secretary-Treasurer: Dr. Frederick H. Roost, 420 Trimble Bldg., Sioux City, Iowa.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. Wiley R. Buffington, 211 Camp St., New Orleans.
Secretary: Dr. W. D. Gill, 323 Medical Arts Bldg., San Antonio, Texas.

SOUTHWESTERN MICHIGAN TRIOLOGICAL SOCIETY

President: Dr. W. E. McGarvey, 802 Jackson City Bank Bldg., Jackson.
Secretary: Don C. Lyon, 1405 Union and Peoples National Bank Bldg., Jackson.
Time: Third Thursday of alternate months.

STATE

Colorado Oto-Laryngological Society

President: Dr. Harry L. Whitaker, 1612 Fremont St., Denver.
Secretary: Dr. Rex L. Murphy, Metropolitan Bldg., Denver.
Place: Assembly Room of Metropolitan Bldg. Time: First Saturday of each month from October to May.

Connecticut State Medical Society, Section on Eye, Ear, Nose and Throat

Chairman: Dr. J. J. Cohane, 59 College St., New Haven.
Secretary-Treasurer: Dr. Walter L. Hogan, 750 Main St., Hartford.

Eye, Ear, Nose and Throat Club of Georgia

President: Dr. Thomas H. Hall, Jr., 617 Mulberry St., Macon.
Secretary-Treasurer: Dr. J. A. Smith, 700 Spring St., Macon.

Indiana Academy of Ophthalmology and Oto-Laryngology

President: Dr. Robert J. Masters, 805 Hume-Mansur Bldg., Indianapolis.
Secretary: Dr. J. Kent Leasure, 611 Hume-Mansur Bldg., Indianapolis.
Place: Indianapolis. Time: Dec. 13, 1933.

Iowa Academy of Ophthalmology and Oto-Laryngology

President: Dr. W. W. Pearson, 410 Sixth Ave., Des Moines.
Secretary-Treasurer: Dr. W. F. Boiler, 105 East Iowa Ave., Iowa City.

Michigan State Medical Society, Section of Ophthalmology and Otolaryngology

Chairman: Dr. Ray Connor, 28 W. Adams Ave., Detroit.
Secretary: Dr. Ralph B. Fast, 136 E. Michigan Ave., Kalamazoo.

Minnesota Academy of Ophthalmology and Oto-Laryngology

President: Dr. Harold J. Rothschild, 239 Lowry Bldg., St. Paul.
Secretary-Treasurer: Dr. W. E. Camp, Medical Arts Bldg., Minneapolis.
Time: Second Friday of each month, October to May.

Montana Academy of Oto-Ophthalmology

President: Dr. Lawrence G. Dunlap, 101 Main St., Anaconda.
Secretary: Dr. A. W. Morse, Phoenix Bldg., Butte.
Place: Anaconda. Time: July 11, 1933.

New Jersey State Medical Society, Section on Ophthalmology, Otology and Rhinolaryngology

Chairman: Dr. Charles H. Schlichter, 556 N. Broad St., Elizabeth.
Secretary: Dr. B. E. Failing, 31 Lincoln Park, Newark.

New York State Medical Society, Eye, Ear, Nose and Throat Section

Chairman: Dr. D. F. Gillette, 109 S. Warren St., Syracuse.
Secretary: Dr. Frank M. Sulzman, 1831 Fifth Ave., Troy.

NORTH DAKOTA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Axel Oftedal, 55½ Broadway, Fargo.
Secretary-Treasurer: Dr. F. L. Wicks, 516 Fourth St., Valley City.

OREGON ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. A. B. Dykman, Medical Dental Bldg., Portland.
Secretary: Dr. Andrew J. Browning, 418 Mayer Bldg., Portland.
Place: Good Samaritan Hospital. Time: Third Tuesday of each month.

RHODE ISLAND OPHTHALMOLOGICAL AND OTOLOGICAL SOCIETY

President: Dr. Herman A. Winkler, 224 Thayer St., Providence.
Secretary: Dr. N. A. Bolotow, 108 Waterman St., Providence.
Place: Rhode Island Medical Library. Time: 8:30 p. m., second Thursday in October, December, February and April.

SOUTH CAROLINA SOCIETY OF OTOLARYNGOLOGY AND OPHTHALMOLOGY

President: Dr. Walter Bristow, Medical Arts Bldg., Columbia.
Secretary: Dr. J. W. Jervey, Jr., 101 Church St., Greenville.
Place: Columbia. Time: October, 1933.

TENNESSEE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. R. G. Reaves, 422 W. Cumberland Ave., Knoxville.
Secretary: Dr. Ralph O. Rychener, 1720 Exchange Bldg., Memphis.
Place: Nashville. Time: April 10, 1933.

TEXAS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. A. F. Clark, 388 Pearl St., Beaumont, Texas.
Secretary: Dr. A. F. Clark, 1034 Nix Bldg., San Antonio.
Place: Dallas. Time: Dec. 1-2, 1933.

UTAH OPHTHALMOLOGICAL SOCIETY

President: Dr. F. M. McHugh, 17 Exchange Pl., Salt Lake City.
Secretary-Treasurer: Dr. W. Leroy Smith, Erza Thompson Bldg., Salt Lake City.

VIRGINIA SOCIETY OF OTOLARYNGOLOGY AND OPHTHALMOLOGY

President: Dr. Frank P. Smart, 142 W. York St., Norfolk.
Secretary-Treasurer: Dr. Henry Grant Preston, Professional Bldg., Harrisonburg.

WEST VIRGINIA STATE MEDICAL ASSOCIATION, EYE, EAR, NOSE AND THROAT SECTION

President: Dr. O. H. Bobbitt, Medical Arts Bldg., Charleston.
Secretary: Dr. Harry V. Thomas, 95 Fairmont Ave., Fairmont.

LOCAL

ACADEMY OF MEDICINE OF NORTHERN NEW JERSEY, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. Elbert A. Curtis, 65 Central St., Newark.
Secretary: Dr. A. Russell Sherman, 671 Broad St., Newark.
Place: 91 Lincoln Park South. Time: 8:45 p. m., second Monday of each month.

ATLANTA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. G. E. Clay, 384 Peachtree St., N. E., Atlanta, Ga.
Secretary: Dr. William O. Martin, Jr., 412 Medical Arts Bldg., Atlanta, Ga.
Place: Academy of Medicine, 38 Prescott St., N. E. Time: 8 p. m., last Thursday of each month.

Baltimore City Medical Society, Section on Otology and Laryngology

President: Dr. James J. Chisolm, Medical Arts Bldg., Baltimore.
Secretary: Dr. Louis Diener, 2449 Eutaw Pl., Baltimore.
Place: 1211 Cathedral St. Time: 8:30 p. m., last Friday of each month.

Buffalo Oto-Laryngological Society

President: Dr. John F. Healey, 925 Delaware Ave., Buffalo.
Secretary-Treasurer: Dr. Herrmann E. Bozer, 468 Delaware Ave., Buffalo.
Place: Buffalo Club. Time: Second Tuesday of each month from September to May, inclusive.

Chicago Laryngological and Otological Society

President: Dr. Charles Younger, 25 E. Washington St., Chicago.
Secretary-Treasurer: Dr. Austin A. Hayden, 25 E. Washington St., Chicago.
Place: Medical and Dental Arts Bldg., 185 N. Wabash Ave. Time: 6 p. m., first Monday of each month from October to May.

Cincinnati Oto-Laryngological Society

President: Dr. Henry M. Goodyear, The Doctors' Bldg., Cincinnati.
Secretary-Treasurer: Dr. Samuel Seltz, Provident Bank Bldg., Cincinnati.
Place: University Club. Time: 6:30 p. m., third Tuesday of each month from October to May.

Cleveland Academy of Medicine, Section of Ophthalmology and Otolaryngology

Chairman: Dr. M. W. Jacoby, 2323 Prospect Ave., Cleveland.
Secretary: Dr. H. C. Rosenberger, 1065 Rose Bldg., Cleveland.
Place: Winton Hotel. Time: Fourth Friday of each month.

College of Physicians of Philadelphia, Section on Otolaryngology

Chairman: Dr. Ralph Butler, 2007 Chestnut St., Philadelphia.
Clerk: Dr. Karl M. Houser, 1826 Pine St., Philadelphia.
Place: College of Physicians. Time: 8:30 p. m., third Wednesday of each month from October to June.

Columbus Ophthalmological and Otolaryngological Society

Chairman: Dr. John B. Alcorn, 167 E. State St., Columbus, Ohio.
Secretary-Treasurer: Dr. Edward W. Harris, 681 E. Broad St., Columbus, Ohio.
Time: First Monday of each month.

Dallas Academy of Ophthalmology and Otolaryngology

President: Dr. O. M. Marchman, 1717 Pacific Ave., Dallas, Texas.
Recording Secretary: Dr. W. Mood Knowles, 1717 Pacific Ave., Dallas, Texas.
Corresponding Secretary: Dr. J. M. Potts, 1717 Pacific Ave., Dallas, Texas.
Place: Dallas Athletic Club. Time: 6:30 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Des Moines Academy of Ophthalmology and Otolaryngology

President: Dr. C. F. Howland, 604 Locust St., Des Moines, Iowa.
Secretary-Treasurer: Dr. Grace Doane, 614 Bankers Trust Bldg., Des Moines, Ia.
Time: Third Monday of each month, September to May, inclusive.

Detroit Oto-Laryngological Society

President: Dr. R. Lee Laird, 513 David Whitney Bldg., Detroit.
Secretary-Treasurer: Dr. J. M. Sutherland, 8-257 General Motors Bldg., Detroit.
Place: Maccabees Bldg. Time: Third Wednesday evening of each month from October to May.

Fort Worth Eye, Ear, Nose and Throat Society

President: Dr. R. H. Needham, 1304 Medical Arts Bldg., Fort Worth, Texas.
Secretary: Dr. Van D. Rathgeber, 1011 Medical Arts Bldg., Fort Worth, Texas.
Place: University Club. Time: 6.30 p. m., first Friday of each month except July and August.

Grand Rapids Eye, Ear, Nose and Throat Society

President: Dr. Carl F. Snapp, Medical Arts Bldg., Grand Rapids, Mich.
Secretary-Treasurer: Dr. Walter W. Oliver, 26 Sheldon Ave., Grand Rapids, Mich.
Place: Various local hospitals. Time: 6:30 p. m., third Thursday of alternating months, September to May.

Houston Academy of Medicine, Eye, Ear, Nose and Throat Section

President: Dr. J. F. Gamble, 201 Main St., Houston, Tex.
Secretary: Dr. B. F. Payne, 1304 Walker Ave., Houston, Tex.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., second Thursday of each month from September to June.

Indianapolis Ophthalmological and Oto-Laryngological Society

President: Dr. John W. Carmack, 23 E. Ohio St., Indianapolis.
Secretary: Dr. Kenneth L. Craft, 23 E. Ohio St., Indianapolis.
Place: Indianapolis Athletic Club. Time: 6:30 p. m., second Thursday of each month from October to May.

Kansas City Society of Ophthalmology and Otolaryngology

President: Dr. Albert N. Lemoine, 906 Grand Ave., Kansas City, Mo.
Secretary: Dr. Harold B. Hedrick, 618 Professional Bldg., Kansas City, Mo.
Time: Third Thursday of each month from September to May. The November, January and March meetings are devoted to clinical work.

Long Beach Eye, Ear, Nose and Throat Society

President: Dr. Ferris L. Arnold, 923 Security Bldg., Long Beach, Calif.
Secretary-Treasurer: Dr. Clyde E. Harner, 314 Security Bldg., Long Beach, Calif.
Place: Professional Bldg. Time: Last Wednesday of each month from September to June.

Los Angeles County Medical Society, Section of Ophthalmology and Otolaryngology

Acting President: Dr. D. E. Godwin, 910 Security Bldg., Long Beach, Calif.
Secretary-Treasurer: Dr. F. H. Brandt, 1052 W. Sixth St., Los Angeles.
Place: Los Angeles County Medical Association, 1925 Wilshire Blvd. Time: 8 p. m., fourth Monday of each month, September to May, inclusive.

Medical Society of the County of Kings, Section on Otolaryngology

Chairman: Dr. Edward Leo Berger, 903 Park Pl., Brooklyn.
Secretary-Treasurer: Dr. Oscar Rodin, 893 Eastern Parkway, Brooklyn.
Place: Library Bldg., County Society, 1313 Bedford St., Brooklyn.
Time: 9 p. m., second Wednesday of January, March, May and November.

Medical Society of the District of Columbia, Section on Ophthalmology and Oto-Laryngology

Chairman: Dr. Albert P. Tibbets, 1801 I St., Washington.
Secretary: Dr. G. Victor Simpson, 1710 Rhode Island Ave., Washington.
Place: 1718 M St., N. W. Time: 8 p. m., third Friday of each month.

Memphis Society of Ophthalmology and Oto-Laryngology

Chairman: Each member, in alphabetical order.
Secretary: Dr. R. O. Rychener, Exchange Bldg., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month.

Milwaukee Oto-Ophthalmic Society

President: Dr. Samuel G. Higgins, 324 E. Wisconsin Ave., Milwaukee.
Secretary-Treasurer: Dr. O. P. Schoofs, 324 E. Wisconsin Ave., Milwaukee.
Place: University Club. Time: 6:30 p. m., third Tuesday of each month.

Nashville Academy of Ophthalmology and Otolaryngology

Chairman: Dr. Eugene Orr, 706 Church St., Nashville.
Secretary-Treasurer: Dr. H. C. Smith, Medical Bldg., Nashville.
Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month, October to June.

New Orleans Ophthalmological and Otolaryngological Society

President: Dr. Charles A. Bahn, 150 Baronne St., New Orleans.
Secretary-Treasurer: Dr. M. F. Meyer, Canal Bank Bldg., New Orleans.
Place: Eye, Ear, Nose and Throat Hospital. Time: January to June and October to January—third Thursday of each month

New York Academy of Medicine, Section of Otolaryngology

Chairman: Dr. Charles Imperatori, 108 E. 38th St., New York.
Secretary: Dr. David H. Jones, 140 E. 54th St., New York.
Place: 103d St. and 5th Ave. Time: 8:30 p. m., third Wednesday of each month from October to May.

Omaha and Council Bluffs Ophthalmological and Otolaryngological Society

President: Dr. L. G. Howard, 532, 1st Ave., Council Bluffs, Iowa.
Secretary-Treasurer: Dr. W. A. Cassidy, 1620 Medical Arts Bldg., Omaha.
Place: Omaha Club, 20th and Douglas Sts., Omaha. Time: Dinner, 6 p. m. Program, 7 p. m., third Wednesday of each month from October to May.

Philadelphia Laryngological Society

President: Dr. Louis H. Clerf, 128 S. Tenth St., Philadelphia.
Secretary: Dr. Herman B. Cohen, 1301 Spruce St., Philadelphia.
Place: College of Physicians. Time: 8:30 p. m., first Tuesday of each month from October to June.

Pittsburgh Otological Society

Chairman: Dr. Roy C. Cooper, 510 S. Aiken Ave., Pittsburgh.
Secretary-Treasurer: Dr. Alexander R. Hampsey, 806 May Bldg., Pittsburgh.

Richmond Ophthalmological and Oto-laryngological Society

President: Dr. R. W. Vaughan, Medical Arts Bldg., Richmond, Va.
Secretary: Dr. William R. Weisiger, Medical Arts Bldg., Richmond, Va.
Place: Westmoreland Club. Time: 6 p. m., second Monday of each month from October to May.

Rochester Eye, Ear, Nose and Throat Society

Chairman: Dr. E. S. Amsler, 53 S. Fitzhugh St., Rochester, N. Y.
Secretary-Treasurer: Dr. E. W. O'Brien, 5 N. Goodman St., Rochester, N. Y.
Place: Rochester Medical Association, 13 Prince St. Time: 8 p. m., third Monday of each month from October to May.

St. Louis Ear, Nose and Throat Club

Acting President: Senior member of the executive committee: Dr. William E. Sauer, 3720 Washington Blvd., St. Louis.
Secretary: Dr. B. J. McMahon, 813 Missouri Bldg., St. Louis.
Place: University Club Bldg. Time: Third Wednesday in February, April, October and December.

San Antonio Ophthalmolo-Oto-Laryngological Society

President: Dr. Charles J. Boehs, 1126 Medical Arts Bldg., San Antonio, Texas.
Secretary-Treasurer: Dr. E. D. Dumas, 425 Medical Arts Bldg., San Antonio, Texas.
Place: Bexar County Medical Library. Time: 8 p. m., first Tuesday of each month from October to May.

San Francisco County Medical Society, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Robert Carson Martin, 384 Post St., San Francisco.
Secretary: Dr. George N. Hosford, 490 Post St., San Francisco.
Place: Society's Building, 2180 Washington St., San Francisco. Time: Third Tuesday of every month except May, June, July and December.

Shreveport Eye, Ear, Nose and Throat Society

President: Dr. T. D. Boaz, Medical Arts Bldg., Shreveport, La.
Secretary-Treasurer: Dr. W. L. Atkins, 940 Margaret Pl., Shreveport, La.
Place: 1240 Texas Ave. Time: 7:30 p. m., first Monday of each month from October to June.

Spokane Academy of Ophthalmology and Otolaryngology

President: Dr. David Hartin, Old National Bank Bldg., Spokane, Wash.
Secretary: Dr. W. W. Henderson, Paulsen Medical and Dental Bldg., Spokane, Wash.
Place: Paulsen Medical and Dental Library. Time: 8 p. m., fourth Tuesday of each month except June, July and August.

Syracuse Eye, Ear, Nose and Throat Society

President: Dr. W. G. Dickinson, 109 S. Warren St., Syracuse, N. Y.
Secretary-Treasurer: Dr. Harold H. Joy, 109 S. Warren St., Syracuse, N. Y.
Place: University Club. Time: Second Friday of each month except June, July and August.

Tulsa Academy of Ophthalmology and Otolaryngology

President: Dr. Roy W. Dunlap, 108 W. 6th St., Tulsa, Okla.
Secretary-Treasurer: Dr. Marvin D. Henley, 108 W. 6th St., Tulsa, Okla.
Place: Medical Arts Bldg. Time: Monthly.